STRENGTH - DURATION CURVES

IN

POLIOMYELITIS

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INTRODUCTION

Poliomyelitis is an infectious disease due to a filterable virus which, it is believed, gains access to the body via the naso-pharynx and the intestine where it multiplies, and to the central nervous system via the autonomic and cranial autonomic nerves (Howe and Bodian 1942). The virus has an affinity for the motor cells of the brain stem and of the anterior horns of the spinal cord, particularly those in the cervical and lumbar enlargements, and the characteristic lesion produced by it is a degeneration of these cells with marked hyperaemia and perivascular lymphocytic infiltration. This results in the principal and crippling symptoms of the disease, muscular paralysis and weakness. Lesions are also found in other parts of the central nervous system and in the posterior root ganglia, and these are thought to be responsible for other features of the disease such as limb pains and neck rigidity.

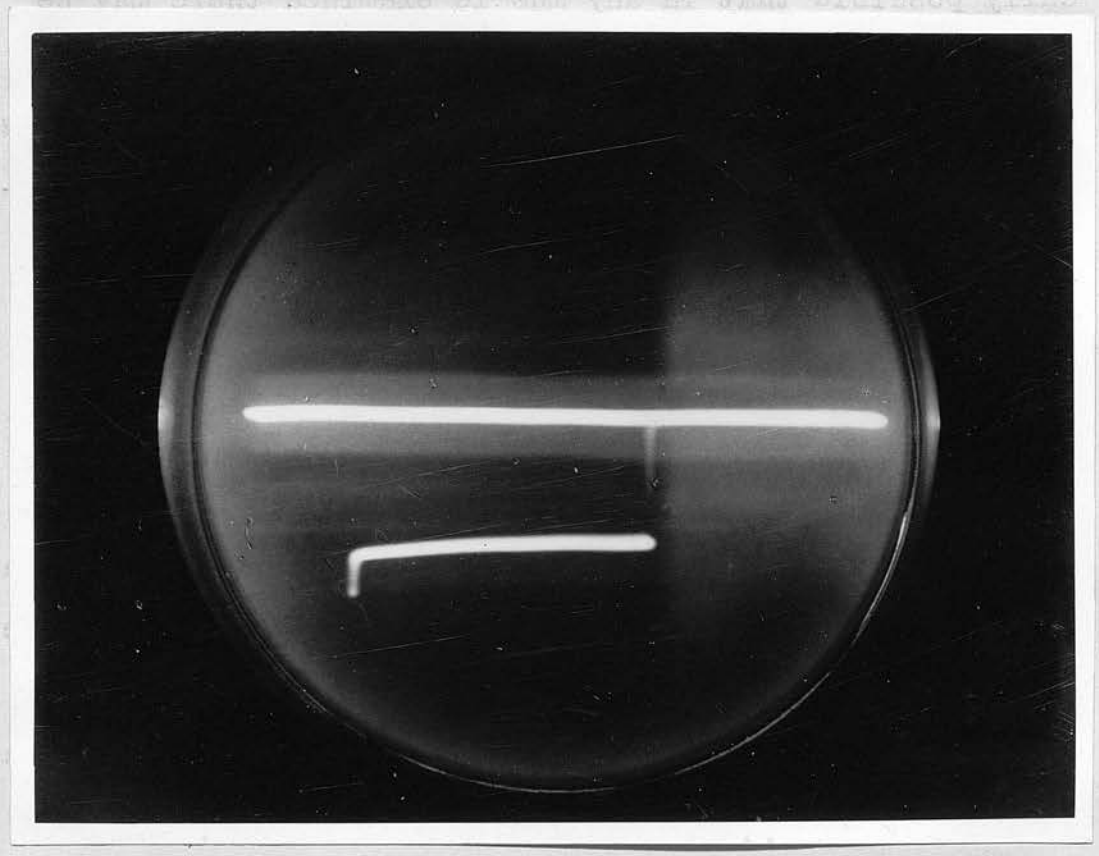
In any condition in which muscle paralysis or weakness is a feature, the electrical reaction of the affected muscles are of interest and may be of importance. These reactions have been extensively studied in peripheral nerve lesions both in the experimental animal and in man (Marble, Hamlin and Watkins 1942; Pollock, Golseth and Arieff 1944,1945(a),(b); Pollock, Golseth, Arieff and

Sherman 1945; Ritchie 1944(a), 1945). After division of a peripheral nerve, conditions are relatively simple since all fibres of any given muscle are usually at the same stage of denervation. In poliomyelitis, however, conditions are much more complicated. It is theoretically possible that in any muscle examined there may be present both normal and denervated muscle fibres, and that the nerve cells connected with the denervated fibres may not all be at the same stage of degeneration. Moreover, when denervated and innervated fibres are present in the same muscle the electrical reactions might be expected to vary depending on which block of fibres is nearer the surface - and therefore the exploring electrode. Yet another complicating factor is that Poliomyelitis is essentially a disease of segmental rather than neural distribution and we do not know to what degree the nerve supply of a muscle is segmental, so that it is possible that one might have a block of denervated fibres with blocks of normal fibres on either side of it with resulting modification of the electrical reactions.

Moldaver (1944) carried out an investigation of the electrical reactions of a group of 51 patients suffering from Poliomyelitis at periods varying from ten days to one year after the onset of the disease, and concluded that all paralysed muscles in Poliomyelitis

Fig.1.

Oscillograph of wave form



Wave 10 M/S duration

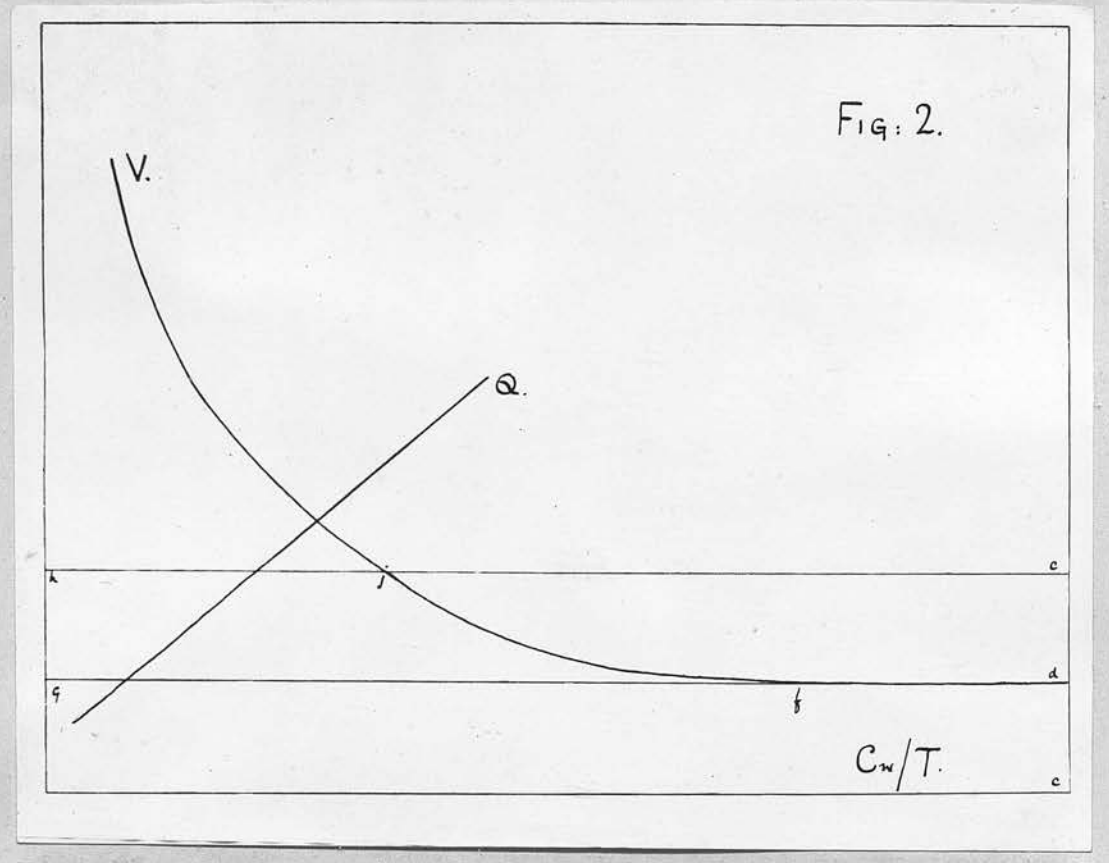
gave electrical reactions characteristic of neuro-muscular degneration. Adrian (1916) had previously shown in a few cases of Poliomyelitis that paralysed muscles show the so-called Reaction of Degeneration. It appears, however, that there is no record of a study of the changes that occur in the electrical reactions done at regular intervals in cases of Poliomyelitis throughout the periods of degeneration, denervation and recovery.

Strength - Duration curves

In order to excite nerve or muscle, a stimulus must be of a sufficient strength and duration, and its rate of change from zero to an effective height must be sufficiently rapid. Using electronic devices, it is possible to devise a stimulator capable of delivering square shaped waves of current of various durations - current which is constant inspite of a variable resistance such as living tissue being included in the circuit (Conrad, Haggard and Teare 1936; Bauwens 1941; Ritchie 1944(b); Walter and Ritchie 1945). These stimulators are of two types - those in which the circuit is arranged so that the current remains constant, and those in which the voltage output remains constant; the results obtained from these two types of stimulator differ quantitatively but are qualitatively similar. The constant-

Fig. 2.

Strength-duration curve and the curve of quantities



V = curve of threshold voltage as a function of the capacity Cw. condenser or of the time T/ (constant current) - the strength-duration curve.

Q = quantity of electricity as a function of Cw/T - the curve of quantities.

c.d. = rheobase.

h. j. = chronaxie.

g. f. = [illegible].

voltage machines have the decided advantage that the stimulus given by them is less painful over the range of voltages employed (Ritchie 1944(b)).

Using such a machine to examine the excitability of a muscle, one has only to consider the strength and the duration of the stimulus since the waves are square in shape - i.e. the rate of change of the stimulus from zero to an effective height is so rapid as to be virtually instantaneous. It is found that if stimuli of varying strengths and durations are applied to a muscle so as to evoke a threshold response in each case, the result is a curve - the so-called Strength-duration curve.

Fig. 2 shows such a curve; from this it will be seen that:- Below a certain strength of stimulus, no response occurs however long the duration; this threshold strength, called the "rheobase" by Lapicque (1926), may be defined as the liminal strength of a stimulus of infinite duration.

Beyond a certain duration of stimulus, further increasing the duration does not lead to a further reduction in the strength required to evoke a contraction; this critical duration of stimulus was named the "temps utile" by Lapicque.

As the duration of stimulus is shortened, so the strength must be increased.

It is difficult to compare the whole of such a curve with others obtained from other patients or the same patient at different times, and it would be convenient to be able to derive from it some index which might be used as a basis for such comparison. The index chosen for this purpose by Lapicque was the "chronaxie" which may be defined as the shortest effective duration of a stimulus of twice rheobasic strength. Lassalle (1928) objected that the chronaxie per se was not an indication of excitability, and suggested another index:-

$$E = a^2 t$$

Where E = excitability

a = rheobase

and t = chronaxie.

This index takes into consideration the height as well as the slope of the curve, and is probably a more logical expression of the condition of the muscle (Walter and Ritchie 1945) but even this is not wholly satisfactory since it is possible to have very different curves with similar Lassalle's indices.

While the strength-duration curve is now generally accepted as being the best means of studying the excitability of a tissue, the story of how this

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While the strength-duration curve is now generally accepted as being the best means of studying the excitability of a tissue, the story of how this

came about is interesting (Fredricq 1928).

Du Bois Reymond (1862) studying the sciatic-gastrocnemius preparation in the frog, showed that in order to be effective a stimulus had to be of a sufficient strength and that the rate of change from zero to its effective height had to be sufficiently rapid; he did not find that the duration of the stimulus played any part in its efficiency. This was generally taught for the next sixty years although in 1863, Fick, working with the Anodonta (River Mussel) in which the muscle is a slower-acting tissue, showed that the stimulus is ineffective if applied for too short a period.

Brucke in 1870 showed that in the muscle of the rabbit's ureter, the threshold rises as the duration diminishes; the following table from his article shows this clearly:-

Intensity (cm. rheocord)	Duration (sec.)
4000	0.25
500	0.5
50	1
25	2
15	3
12	4
11	5
10.5	6

These observations showed that the duration of the stimulus was obviously important, and in 1892, Hoorweg, using condenser discharges, demonstrated this in the

same type of nerve-muscle preparation that had been used by Du Bois Reymond. With condenser discharges and with constant voltage, the duration of the active discharge varies with the capacity of the condenser; Hoorweg showed that as the capacity decreases, so is it necessary to increase the voltage in order to obtain a response.

Weiss in 1901 carried out a series of similar experiments on motor nerves in the frog; he used the constant current (make) stimulus which has the advantage over condenser discharges that it gives a square shaped wave instead of an exponentially-decaying one. His results enabled him to construct a curve of quantities of electricity required to stimulate; this is a straight line whose origin is a positive value on the axis of ordinates of the curve of voltage expressed as a function of time - which is a branch of an equilateral hyperbola. This is shown in Fig. 2. For long durations of current, the threshold current remains the same, and Du Bois Reymond's observations were correct; at shorter durations, however, the threshold rises as the duration diminishes, and Du Bois Reymond's observations were incorrect for this part of the curve. This figure also shows the rheobase, chronaxie, and temps utile of Lapicque.

(c.d.) (h.j.) (g.f.)

Outline of the investigation

Pollock (1945(a)) has laid down a set of conditions which should be complied with by those studying strength-duration curves:-

The apparatus should be so constructed that changes in resistance of the tissues should not modify the current passing through them;

Square wave stimuli should be used.

The current should be measureable.

The durations should be measureable.

The duration of the stimuli should include one of as long as 100 milliseconds.

The size and position of the electrodes should be described.

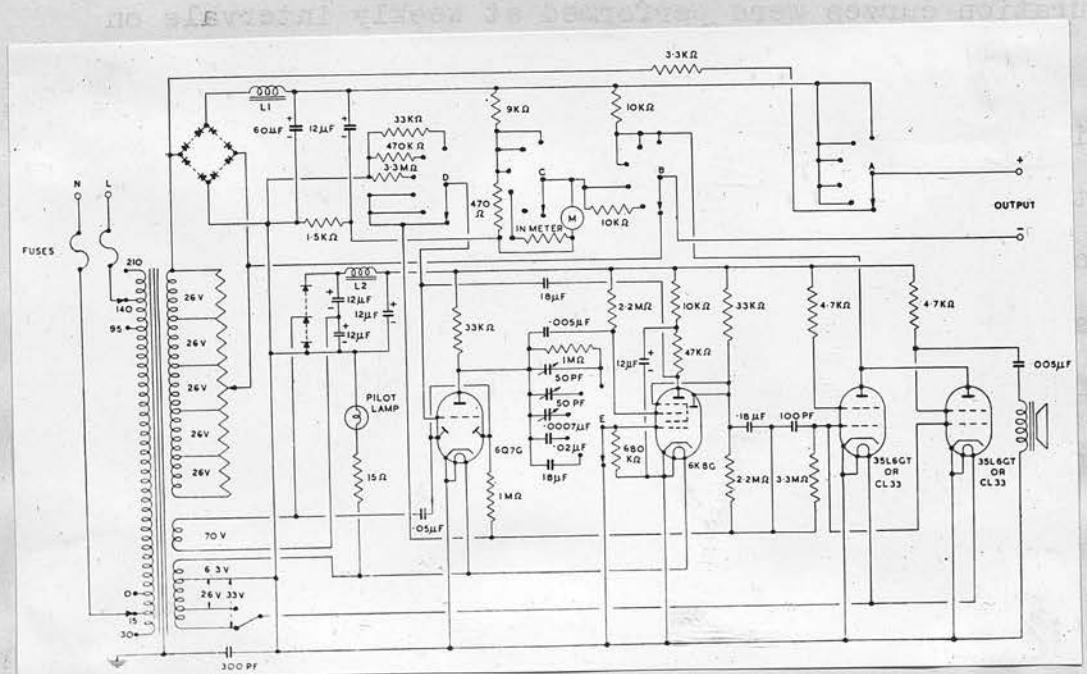
Fulfilling these conditions, strength-duration curves were performed at weekly intervals on a series of 30 recent cases of Poliomyelitis; variations in the characteristics of the curve were noted at various stages of the disease together with the voluntary power of the muscle examined. An attempt was made to correlate these findings with the course of the disease and to indicate how the strength-duration curve may be used as a guide to prognosis and treatment.

Fig. 3.

The Ritchie stimulator

Fig. 3b.

Circuit diagram



EXPERIMENTAL METHOD

The apparatus used for this study (Fig.3 & 3B), was that devised by Walter and Ritchie (1944(b), 1945). This enables one to obtain a stimulus of rectangular (Fig. 1) wave shape at the following seven different durations - 100, 10, 1, 0.5, 0.1, 0.05, 0.02 milliseconds. This machine is of the constant voltage type.

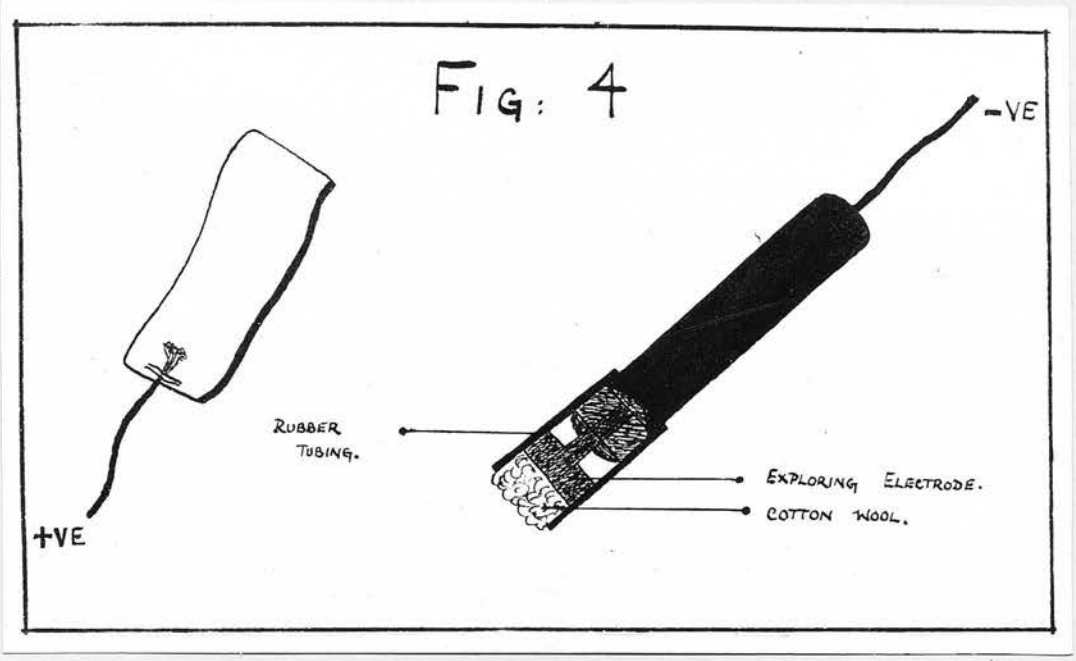
The material consisted of 30 cases of Poliomyelitis whose ages ranged from three weeks to 34 years; apparently normal muscles, as well as those completely paralysed, and some which were partially paralysed, were examined in these cases. Five cases of peripheral nerve injury, three of spastic paralysis, and three normal subjects, were also examined as controls. The examinations on the cases of Poliomyelitis were repeated from as early as three weeks to as late as six months after the onset of the disease.

The examinations were carried out as far as possible under standard conditions, being done in a warm room in good lighting with the muscle under examination in a relaxed position.

The indifferent electrode measuring 23 x 7.5 cm, consisted of a flexible lead plate, and was covered with a detachable cover of felt; it was placed at a convenient

Fig. 4.

Diagram of electrodes



place round the limb being examined. The exploring electrode, the cathode, measured 1 cm. in diameter, and was surrounded at its end by a piece of rubber tubing 2.5 cm in length and 1 cm. in internal diameter; this projected slightly beyond the end of the electrode, and the interval was filled with cotton wool; this was placed over the motor point of the muscle being examined. At the first examination, the motor point was found by exploring with the electrode until the point giving the maximum contraction at a constant voltage was found. A denervated muscle, by definition, has no motor point, but the optimum point for stimulation was found in the same way. Once the exploring electrode had been placed in position, it was not moved during the rest of the examination, and when this had been completed, its site was marked with an indian ink tattoo mark so that the same position of the electrode was ensured at subsequent examinations.

Before use, both electrodes were soaked in a buffer solution composed of:-

$Na_2 H.PO_4$ 25/grams
$Na.H_2 PO_4$ 12/grams
Water to one litre.

: diluted 1/10 before use, and the skin over the motor point of the muscle was moistened with the same solution. (Guttmann, unpublished).

The rubber tubing and cotton wool over the end of the exploring electrode had the effect of making it adaptable to any surface on which it was placed, and the buffer solution was intended to ensure a perfect contact and to reduce electrolytic changes and cutaneous resistance.

The frequency of the stimuli was kept constant at 0.75 cycles/sec. (our machine having been modified to halve the lowest rate of stimulation on the standard model as it was found that this facilitated the accurate determination of the end point). The results were plotted on graphs where the ordinate represented the voltage and the abscissa the duration of the stimulus, the scale of the latter being logarithmic. The rheobase was taken as the threshold at 100 milliseconds (although from some of the curves it is clear that this is not the true rheobase) and the chronaxie was read from the graph as the point where it crossed the voltage line representing twice the rheobase. Lassalle's index was calculated from these figures. The shape of the curve, and the number of the seven different durations of stimulus that were effective were noted.

At the same time as the strength-duration curve was done, the voluntary power of the muscle examined was estimated; this was recorded using the

system recommended by the Peripheral Nerve Injuries Committee of the Medical Research Council:-

5 = contraction against gravity and powerful resistance.

4 = contraction against gravity and some resistance.

3 = contraction against gravity.

2 = movement of the limb with gravity eliminated.

1 = flicker of contraction only.

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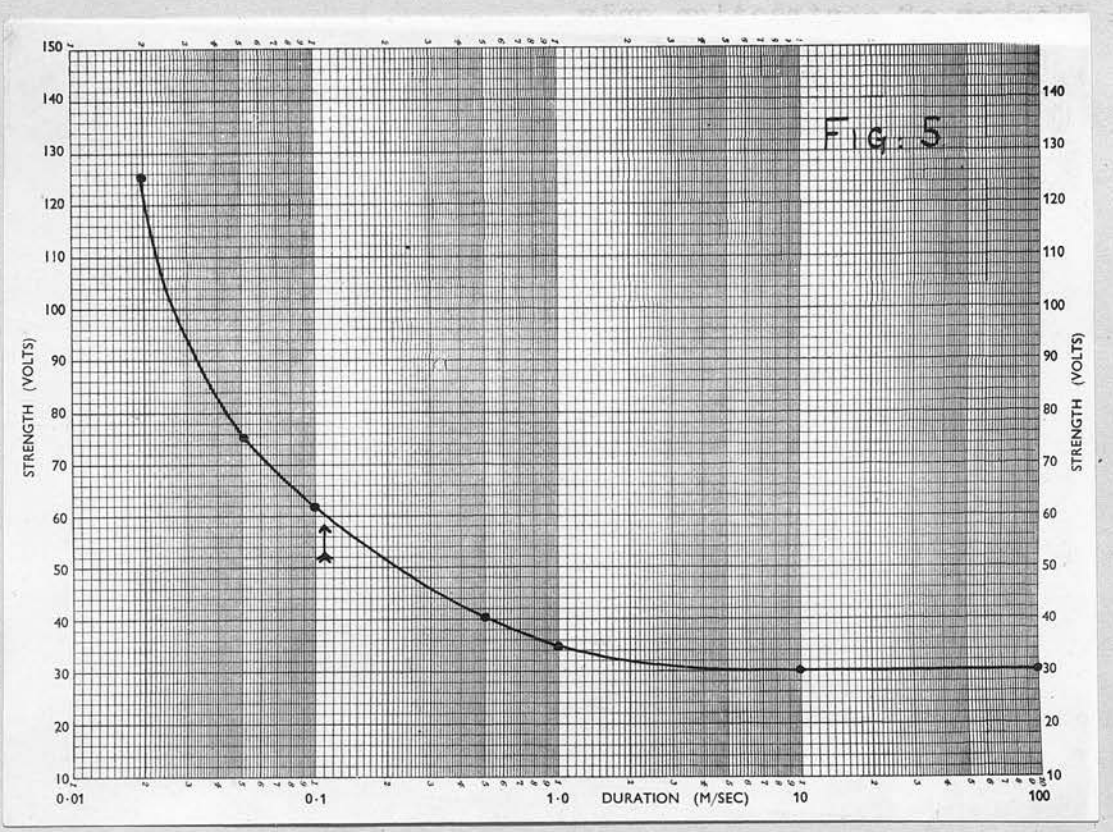
Fig. 5.

Normal strength-duration curve



INTERPRETATION OF RESULTS

In order to view the experimental results in their true perspective, it is necessary to have some knowledge of the strength-duration curves which characterise normal and denervated muscle, and of the changes that have been described in the curves during degeneration and recovery in peripheral nerve lesions.

Characteristics of a normal strength-duration curve

Fig. 5 shows the curve from the right tibialis anterior of a normal subject. In using it as a basis for comparison with other curves, several factors should be taken into consideration.

The shape is smooth; the rheobase is relatively low (30), and in this region the curve is flat. Beyond five milliseconds ("temps utile" of Lapicque) lengthening the duration of the stimulus does not result in a lowering of its threshold. At the shorter durations of stimulus the curve rises more steeply; it is convenient to take the threshold at 0.5 milliseconds as an index of this rise, and in the normal curve it is (40), but little higher than the rheobase. The slope varies in different parts of the curve; the threshold at 100 and 0.5 milliseconds giving some indication of its steepness. A further indication is

Fig. 6.

Complex curve - double in form

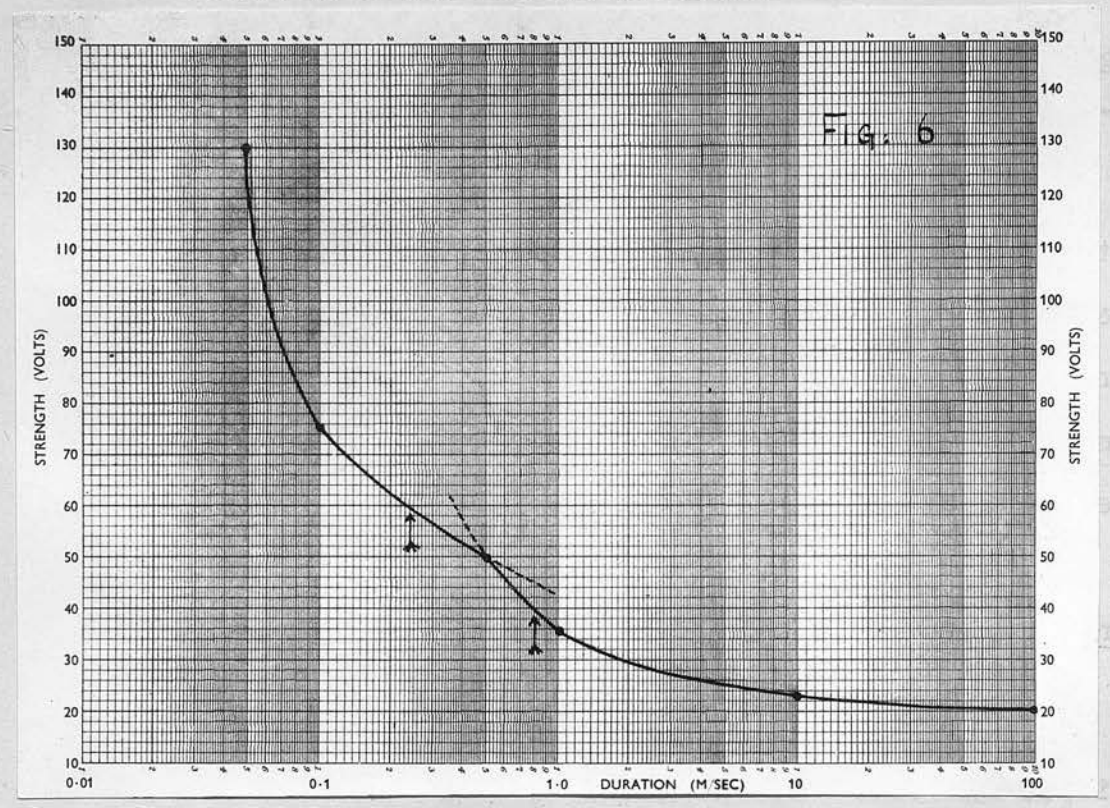
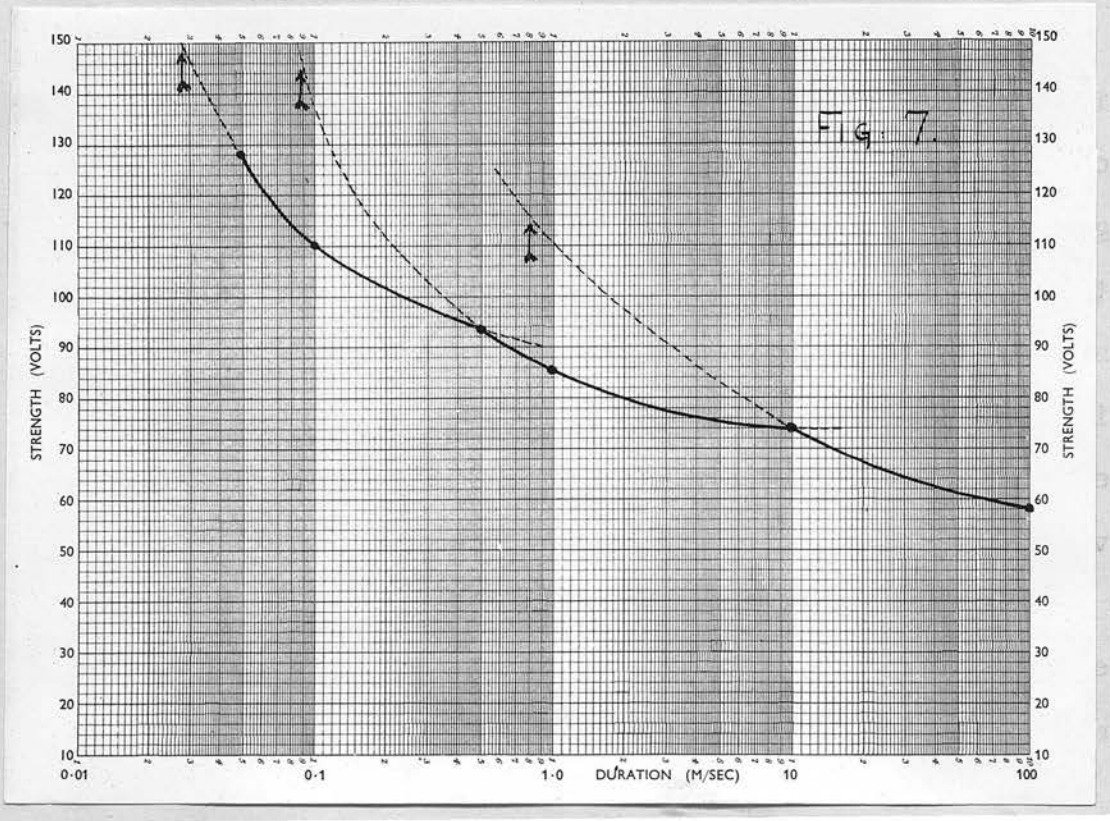


Fig. 7.

Complex curve - triple in form



given by the chronaxie which in normal muscle is low (0.11). Lassalle's index (R^2 x C) is also relatively low (990) in normal muscle. Under normal conditions, the muscle responds to all seven durations of stimulus, and the number of effective stimuli is a very rough indication of the excitability of a muscle.

Two other points are worth mention in a consideration of the normal strength-duration curve - they are incapable of exact measurement or graphic representation and are thus of limited value - the examination of a normal muscle is a painless experience, and the contraction of a normal muscle is a rapid twitch.

Variations that occur in the shape of the curve

Fig.6 shows the curve obtained from the partially paralysed (voluntary power = $2\frac{1}{2}$) right tibialis anterior of a moderately severe case of Poliomyelitis six weeks after the onset of the disease. The stimulus of 0.02 milliseconds duration was ineffective; it is not possible to join up the remaining six points in one smooth curve, and when they are joined a discontinuity is apparent at 0.5 milliseconds.

Fig. 7 shows the curve obtained from the completely paralysed right tibialis anterior of a case six weeks after onset, the week before a flicker

denoting recovery was noted. Again, the stimulus of 0.02 M/S duration was ineffective; it is not possible to join up the remaining six points in even two smooth curves, and when they are joined, discontinuities are evident at 0.5 and 10 M/S.

These variations in shape are representative of numerous similar variations which have been observed in this series. Their significance is discussed more fully later, and it is sufficient for the moment to note that Pollock(1944) has demonstrated similar discontinuities in the strength-duration curves of cats following section and suture of the sciatic nerves; he states that they occur during denervation and recovery, but are not found in normal or completely denervated muscle.

Pollock (1944,1945) worked with current-duration curves and not voltage-duration curves as have been used in this series, but the shape of the two types of curve is similar and although the indices derived from them are not quantitatively comparable, the changes that occur in them during denervation and recovery should be qualitatively comparable (Bauwens 1943).

Variations that occur in the height of the curve

The height of the strength-duration curve varies during denervation and recovery, and the varia-

tions are not of the same order for different durations of stimulus; by observing the changes that occur in the thresholds at 100 and 0.5 milliseconds, one can get some idea of the changes that occur in the height of the curve as a whole and its various parts.

Pollock (1945(a)) has investigated these changes in the constant current curves of the gastrocnemius of experimentally-produced sciatic nerve lesions in the cat. He found that during degeneration there is a temporary early rise of threshold for stimuli of all durations occurring six to fourteen days after nerve section and lasting a few days only before falling again; there is then a second rise which may be continuous or which may consist of two separate peaks, and this is followed by a fall for long duration stimuli coincident with a rise for stimuli of short duration.

In the denervated state, there is, according to Pollock, a continued fall for long duration stimuli, the threshold for which is lower in denervated than in normal muscle. This lowering of the rheobase in the denervated state has not been evident in this series, and we have not met it in the cases of peripheral nerve injury that we have examined. We have, however, found, and the point is emphasised later, that in early stages of Poliomyelitis - four weeks after

onset - one of the distinguishing features of the curves of paralysed muscles which later recover from those of muscles which do not recover is that the rheobase of the latter is much lower than that of the former.

At about the time when voluntary power first returns there is a third rise of threshold for stimuli of all durations, and during the phase of recovery the high threshold persists for stimuli of long duration, but falls for stimuli of short duration.

Curves from this series of cases show somewhat similar changes as these described by Pollock(1945); we have also found that environmental factors affect the height of the strength-duration curve particularly at the long durations of stimulus. These findings are illustrated in Figs. 14-19 (p.29-32).

Variations that occur in the slope of the curve

Adrian (1916) showed in a case of facial palsy that the chronaxie rose from 0.24 M/S in the normal muscle to 10 M/S in the denervated muscle. Pollock (1945(b)) has investigated the evolution of this change both in the experimental animal and in man. He found that during degneration there is an early rise and fall which is followed by a second rise. Denervated muscle

has a higher chronaxie (8). Recovery is indicated by a rapid and considerable fall which follows on a progressive rise to a peak.

As Ritchie (1945), quoting Grey Walter, points out, the actual figures obtained for chronaxie varied with the method of examination used, but our results have shown the same types of change as those described by Pollock (1945).

Table 1 (p.25) shows the figures for chronaxie given by Bourguignon (1923) and shows how the chronaxie varies in different muscle groups.

We have found that the chronaxie also varies slightly with environmental factors. This is shown in Figs.14 - 19 (p.29 - 32).

Table 2 (p.25) shows the figures for chronaxie given by Moldaver (1944) and the differences between this and Table 1 illustrates how the value of this index varies with the experimental method used.

No description could be found of the changes that occur in Lassalle's index during degeneration and recovery but in our experience, they are very similar to those in chronaxie.

TABLE 1

		Chronaxie m/s
Arm:	Flexors	0.08 - 0.16
	Extensors	0.16 - 0.32
Forearm:	Flexors and pronators	0.20 - 0.36
	Extensors and supinators	0.44 - 0.72
Thigh:	Hip flexors	0.10 - 0.16
	Hip extensors	0.44 - 0.72
	Knee extensors	0.44 - 0.72
	Ham strings	0.44 - 0.72
Leg:	Anterior tibial group	0.24 - 0.36
	Calf	0.44 - 0.72

TABLE 2

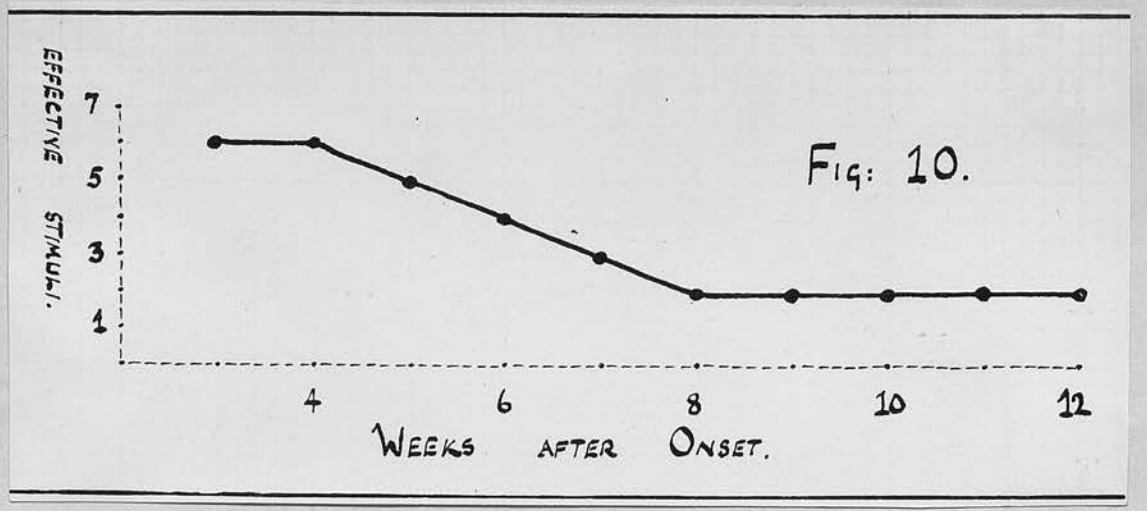
Muscles	Chronaxie m/s
Quadriceps	0.06 - 0.16
Hamstrings	0.16 - 0.32
Tibialis anterior	0.16 - 0.32
Peronei	0.16 - 0.32
Calf	0.16 - 0.32
Orbicularis oris	0.16 - 0.32
Spinati	0.16 - 0.32
Pectoralis major	0.08 - 0.16
Triceps	0.16 - 0.32
Deltoid	0.06 - 0.16
Biceps	0.06 - 0.16
Opponens pollicis	0.16 - 0.32

Variations that occur in the length of the curve

Fig.10 (p.26) shows the graph obtained by plotting the number of effective stimuli at weekly intervals in a severe case in which both lower limbs were completely and permanently paralysed; the muscle examined

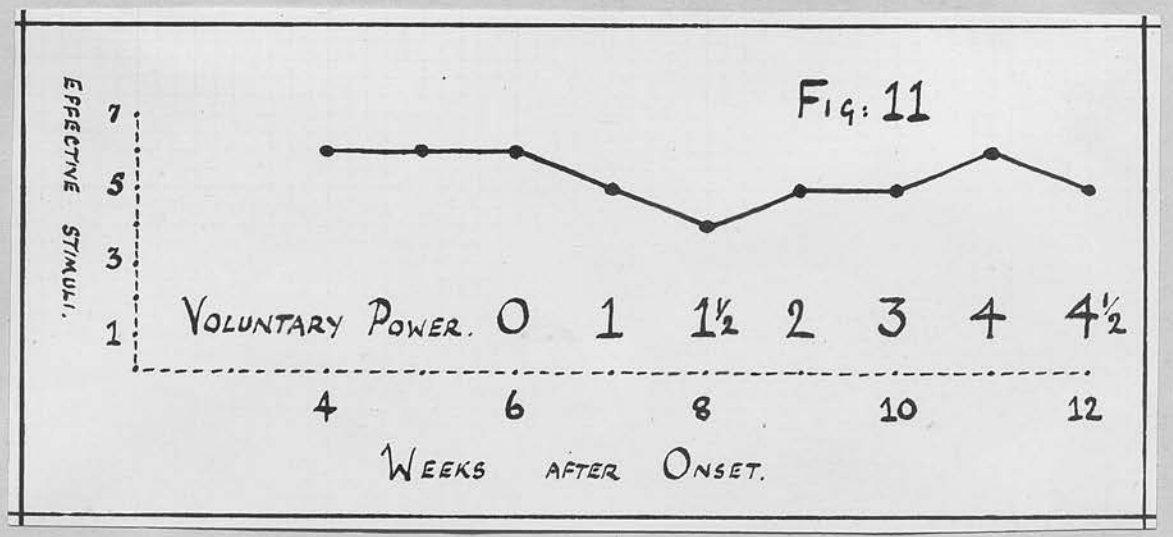
The length of the S/D curve in paralysed muscle.

Fig.10.



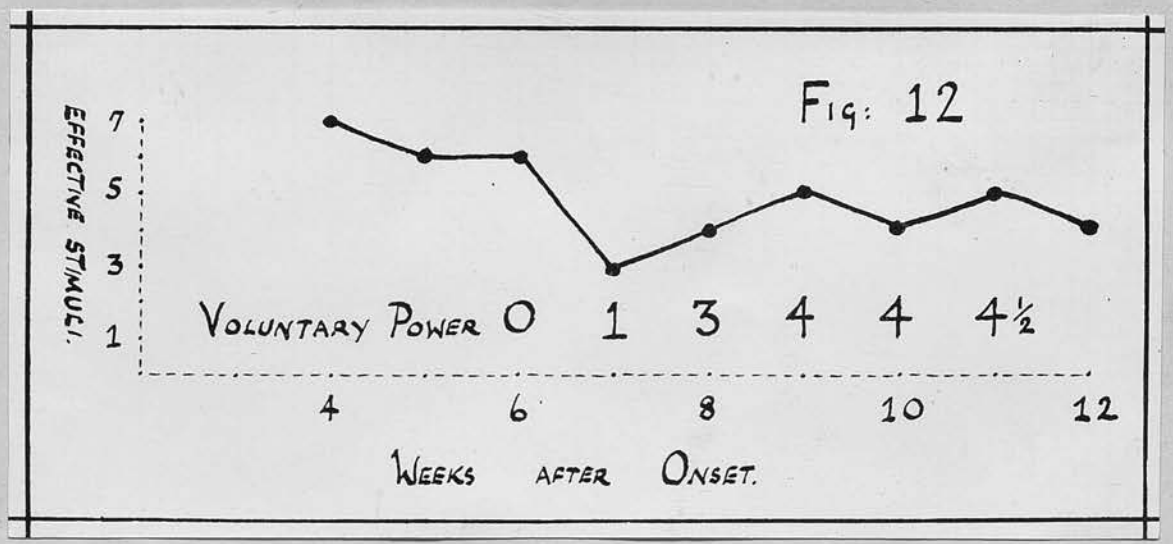
The length of the S/D curve in recovering muscle

Fig.11.



The length of the S/D curve in recovering muscle

Fig.12.



Note in Fig.11 and Fig.12 the falling off of number of effective stimuli with return of voluntary power.

was the left quadriceps. The graph shows how the number of effective stimuli diminishes from the 4th to the 7th week and then remains stationary, the only effective stimuli being those of 100 and 10 M/S duration.

Figs.11 and 12 show similar graphs from both tibialis anterior of another case in which there was recovery; they show how the number of effective stimuli begins to fall just before the return of voluntary power, and then rises again towards normal as recovery proceeds. There is a second slight fall in one of these graphs coincidental with the beginning of walking. The normal value of seven was not reached within the six months during which these cases have been observed.

Other variations which may occur

The examination was usually painless, but on a few occasions was far otherwise; in most cases where this occurred it was associated with a rather higher voltage than usual being required for stimulation, but a few individuals complained of pain even at relatively low voltages; without some objective method of measuring pain it is difficult to come to any definite conclusion on this matter, but the cases in which pain on stimulation was a factor were those cases in which other painful phenomena, such as local muscle tenderness or pain on muscle stretching were observed.

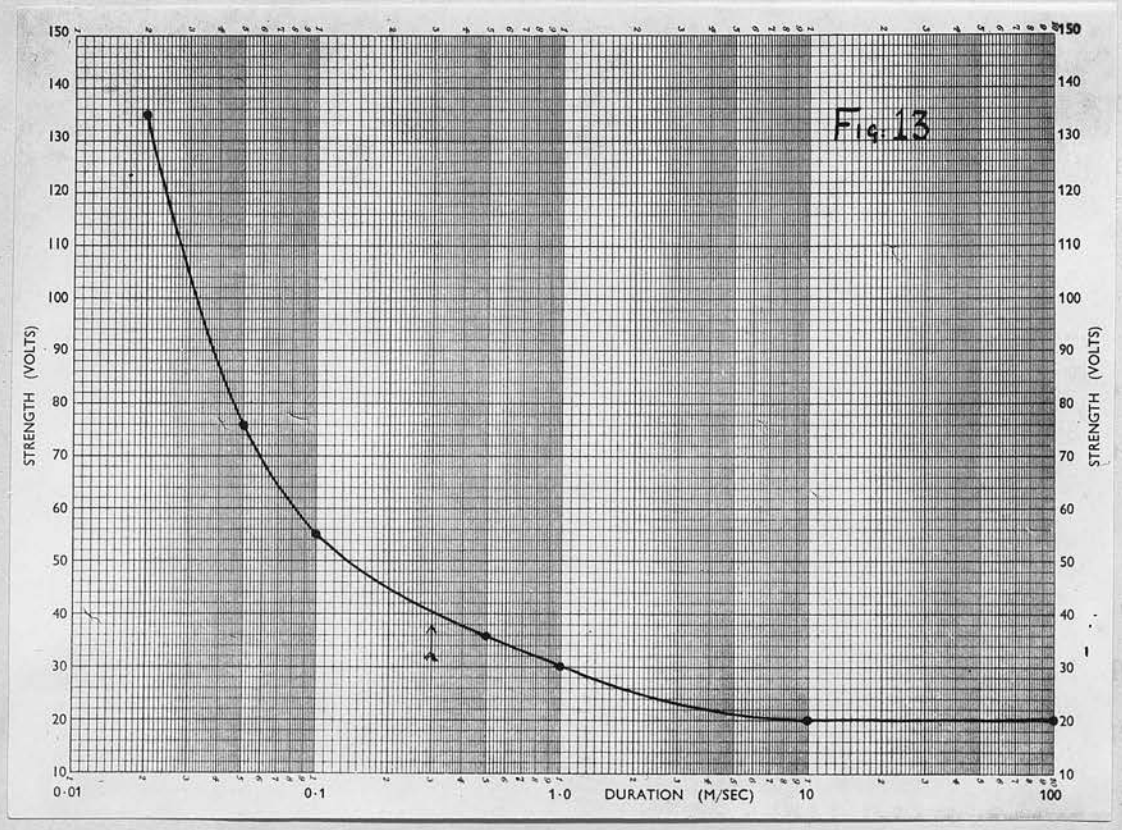
The response of a normal muscle to electrical stimulation is rapid twitch, and during the early stages of denervation (three to five weeks after the onset of paralysis) the response appears rapid. In cases that did not recover, as time went on (13 to 15 weeks after onset) the response became markedly sluggish. Although it is easy to recognise the two extremes in speed of contraction, intermediate stages are impossible to evaluate without some exact method of measurement.

This question of character of muscle response is further discussed later in relation to discontinuities in the strength-duration curve.

Nerve stimulation was carried out as part of the examination in a certain number of the cases; while in general it was not found that this method of examination yielded results that were of much value, it was noticed on one occasion that there was a response from stimulation of the nerve in a muscle which was clinically completely paralysed. This anomaly might be explained by supposing that a synchronous contraction of the few remaining intact motor units, in response to stimulation of the motor nerve, was sufficient to produce a visible twitch, where their asynchronous volitional contraction were insufficient to appreciate clinically.

Fig. 13.

The S/D curve of unaffected muscle in Poliomyelitis



This curve is within normal limits

Variations due to environmental factors

Fig.13 shows the curve obtained from the right tibialis anterior of a case of Poliomyelitis four weeks after the onset; in this case there was only slight paresis of the left lower limb and the right side was clinically normal. The characteristics of the curve are slightly different from those of the curve of normal muscle illustrated in Fig.5. The rheobase is 20, the threshold at 0.5 M/S 36, the chronaxie is 0.3 and Lassalle's index is 120.

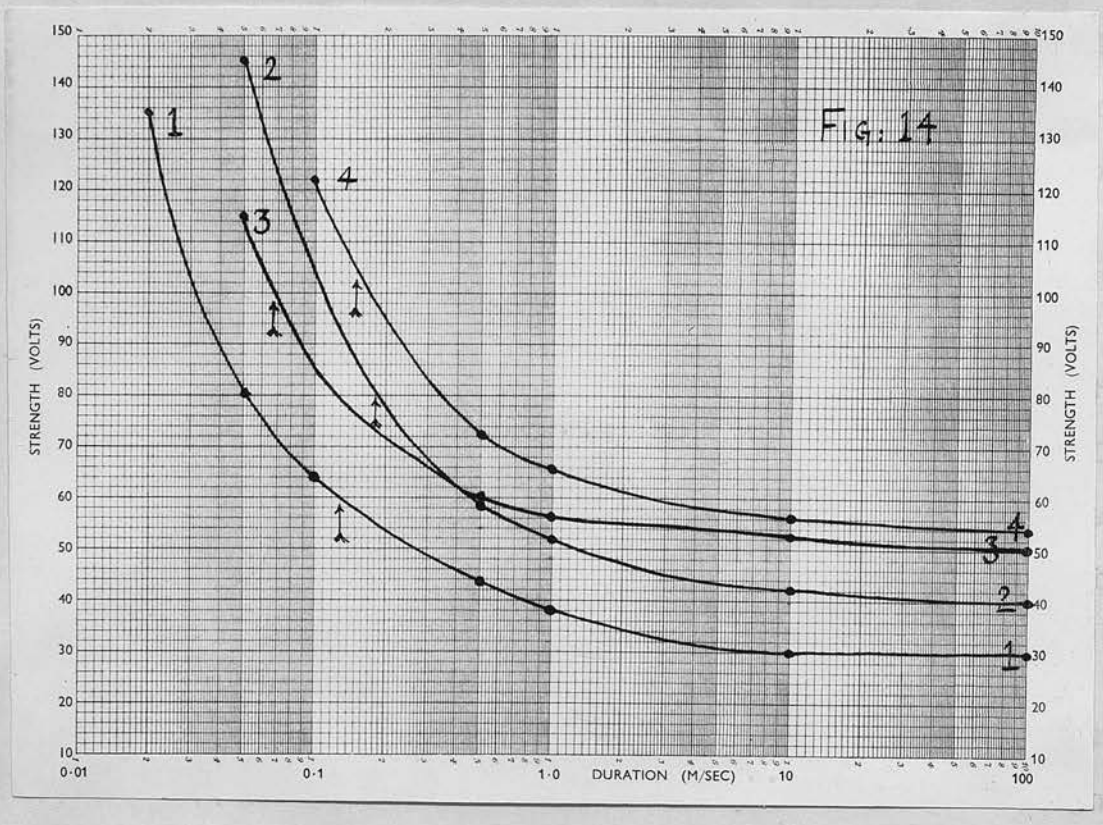
The strength-duration curve obtained from normal muscle however, varies slightly from day to day, and on one occasion a curve exactly similar to that shown in Fig.13 was obtained from the same normal muscle whose curve recorded another day is shown in Fig.5 (p.19).

This leads one to conclude that the unaffected muscles in Poliomyelitis give a normal strength-duration curve, and that the curve obtained from normal muscle varies slightly from day to day.

A series of short experiments were undertaken to determine roughly what order of variation could be expected in the strength-duration curve of muscles under different conditions, and which characteristics of the curve varied the most.

Fig. 14.

The effect of oedema on the S/D curve



These variations have been emphasised by previous writers who stress the need for constancy in minor points of technique if one is to obtain results of any value from the electrical method of examination.

Ritchie (1945) has drawn attention to the marked rise in the value of the rheobase that occurs as the exploring electrode is moved away from the motor point of the muscle (See Fig.50,p.57). Bauwens (1943) stressed the effects of oedema, temperature, and increased blood supply on the strength-duration curve. Grundfest (1932) using a single nerve fibre muscle preparation showed very beautifully how the chronaxie of both nerve and muscle could be varied by using different sized electrodes, how that of muscle varied with the solution with which it was perfused, and how that of nerve varied with the size of the fibre examined.

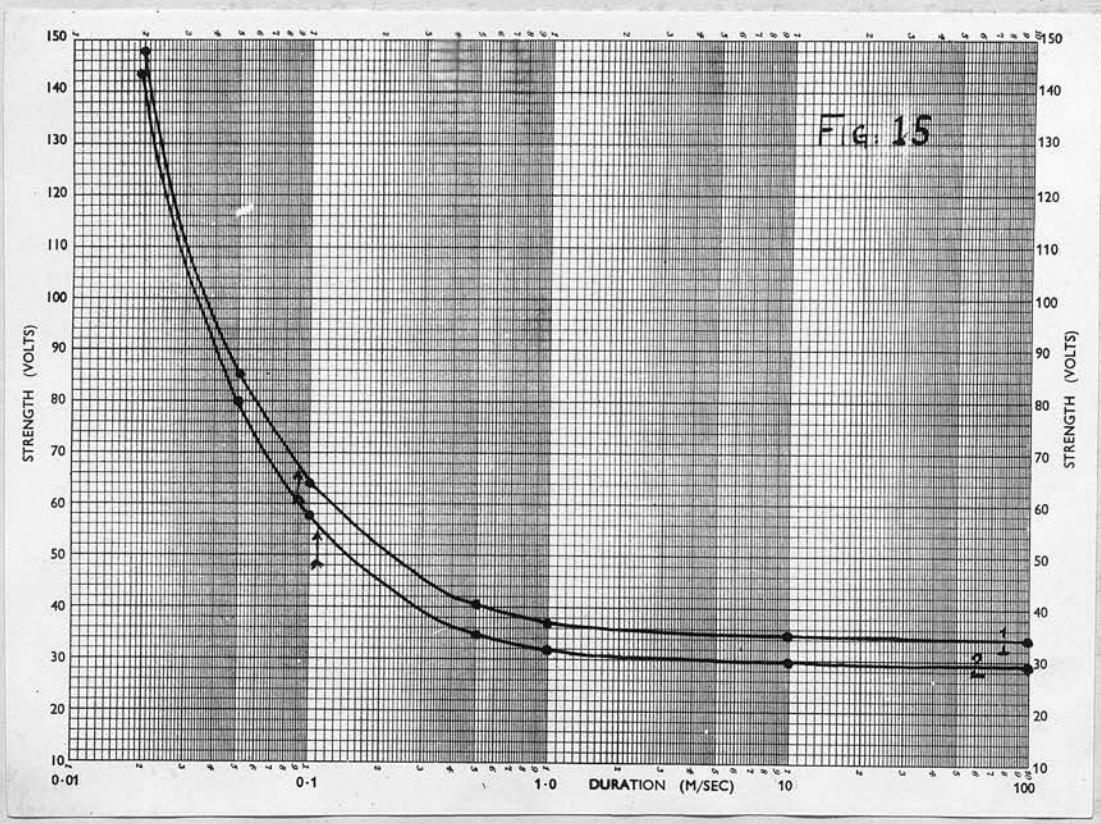
Fig.14 shows the effects of "oedema" - saline injected subcutaneously and intramuscularly around the motor point of the muscle - in the normal subject; the tibialis anterior was used for this experiment.

No.1 shows the original curve.

2 shows the curve after injecting 5 cc normal saline subcutaneously around the motor point.

Fig. 15.

The effect of exercise on the S/D curve



No.3 shows the curve after injecting a further 5 cc intramuscularly around the motor point.

4 shows the curve after injecting a further 10 cc fluid, half subcutaneously and half intramuscularly; the determination of curve No.3 was painful at the long durations of stimulus, and the fluid used for No.4 was 2½ per cent planocaine, which relieved the pain.

Putting on pressure was only demonstrable after the final injection; that is to say that 13 cc fluid had been injected into the limited area around the motor point without being clinically obvious. This suggests that this cause of variation in the strength-duration curve may be more common than one supposes.

Fig.15 shows the effect of exercise - against two 50 lb. springs for a period of 15 minutes - in the normal subject; the muscle examined was again the tibialis anterior.

No.1 shows the curve before exercise.

2 shows the curve immediately after.

The curve was repeated after resting for ten minutes, and the result was identical (within two volts either way) with No.1.

Fig.16 shows the effects of galvanism on a paralysed muscle; the muscle examined was the tibialis

Fig. 16.

The effect of galvanism on the S/D curve in paralysed muscle

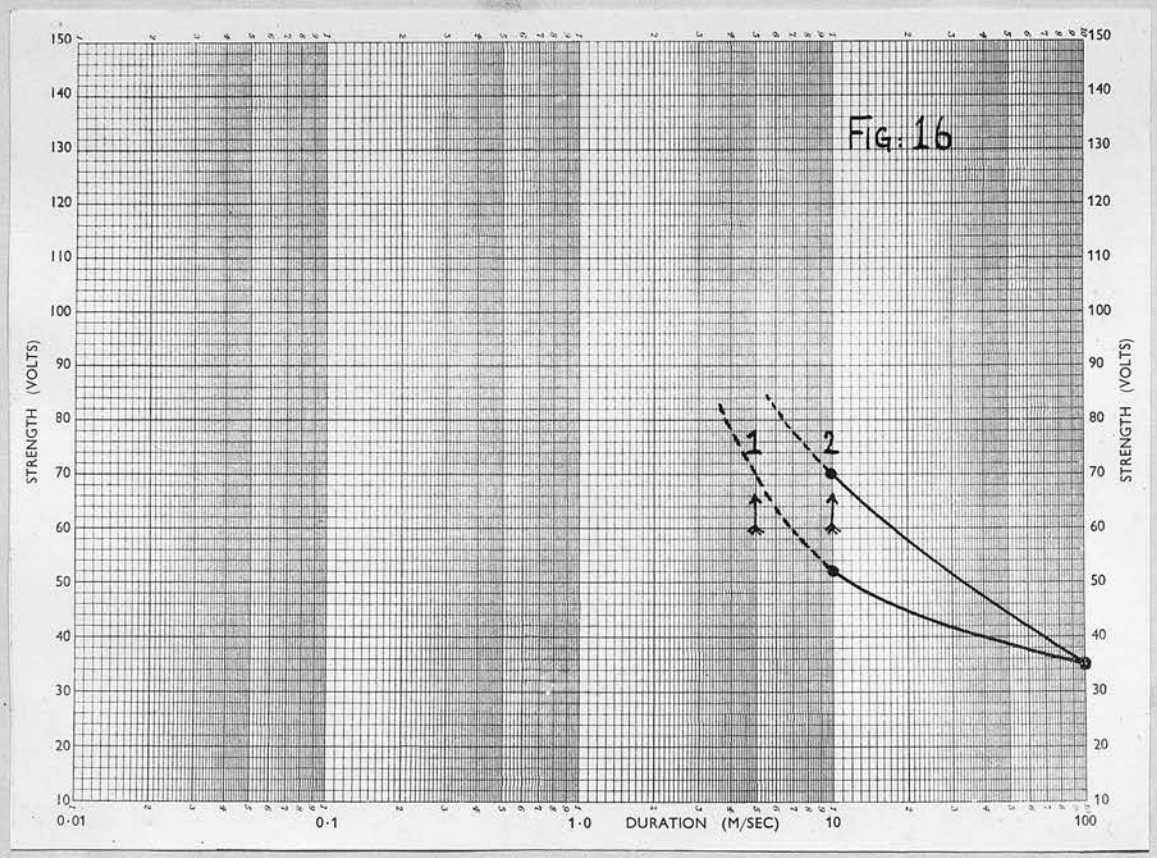
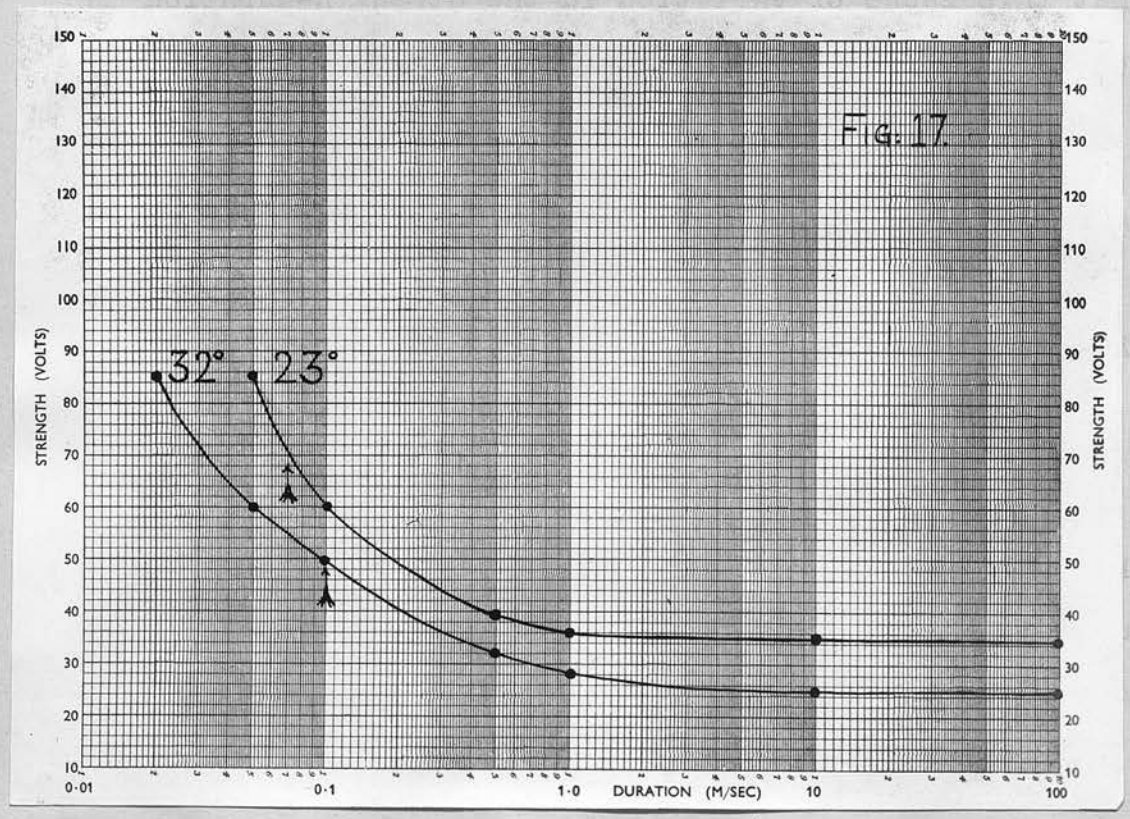


Fig. 17.

The effect of temperature on the S/D curve



Figures refer to skin temperature.

anterior, and interrupted galvanism was given for a period of 15 minutes.

No.1 shows the curve before.

2 shows the curve after.

It will be noted that although the chronaxie was altered the rheobase did not vary in this case as in the others.

Fig. 17 shows the effects of temperature on the strength-duration curve of the tibialis anterior, of a normal subject. The curves were recorded when the skin temperatures were stable, first after heating the subject in a heating box, and then after exposing him in a cool room with the windows and doors open.

Fig.18 shows the effect of occluding the venous return - for a period of 15 minutes with a sphygmomanometer cuff blown up just above the venous pressure - on the strength duration curve of the first dorsal interosseus of a normal subject.

No.1 the resting curve

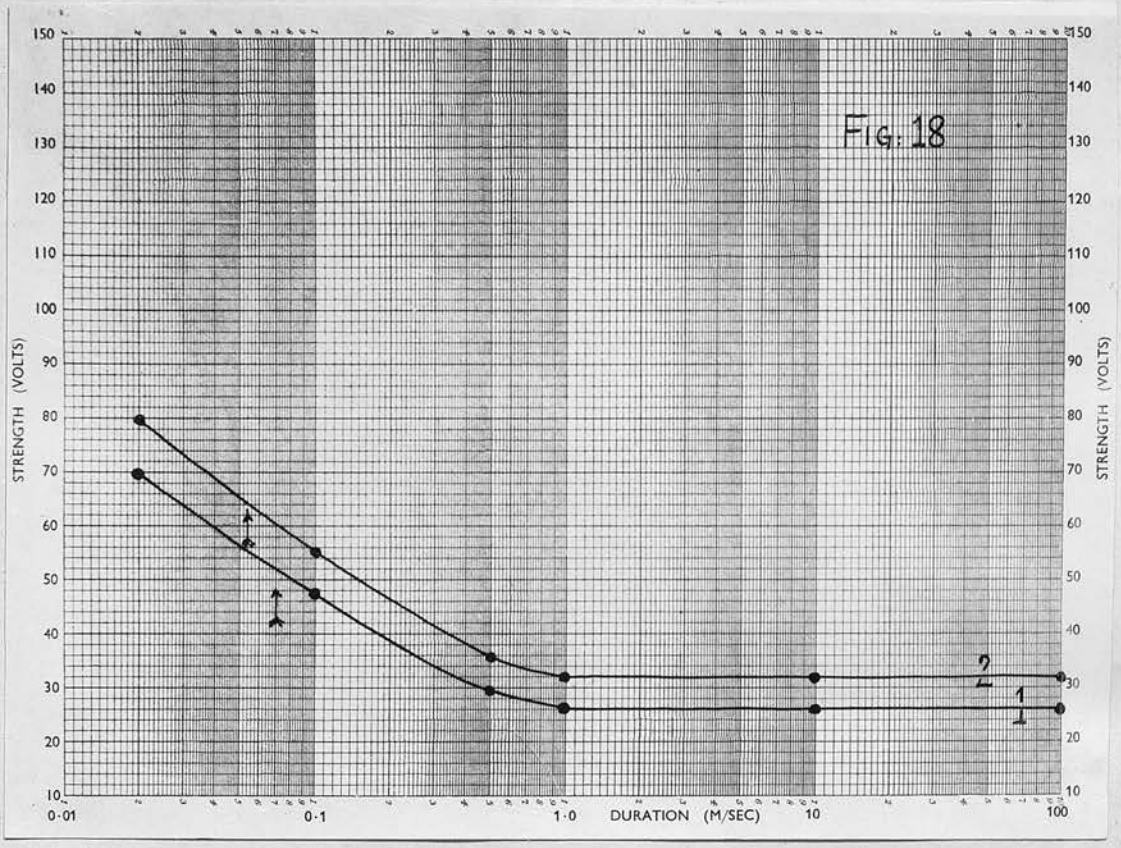
2 the curve after ten minutes occlusion.

Fig. 19 shows the effects of diminished blood supply on the curve of the tibialis anterior of the normal subject.

No.1 shows the curve in the resting state.

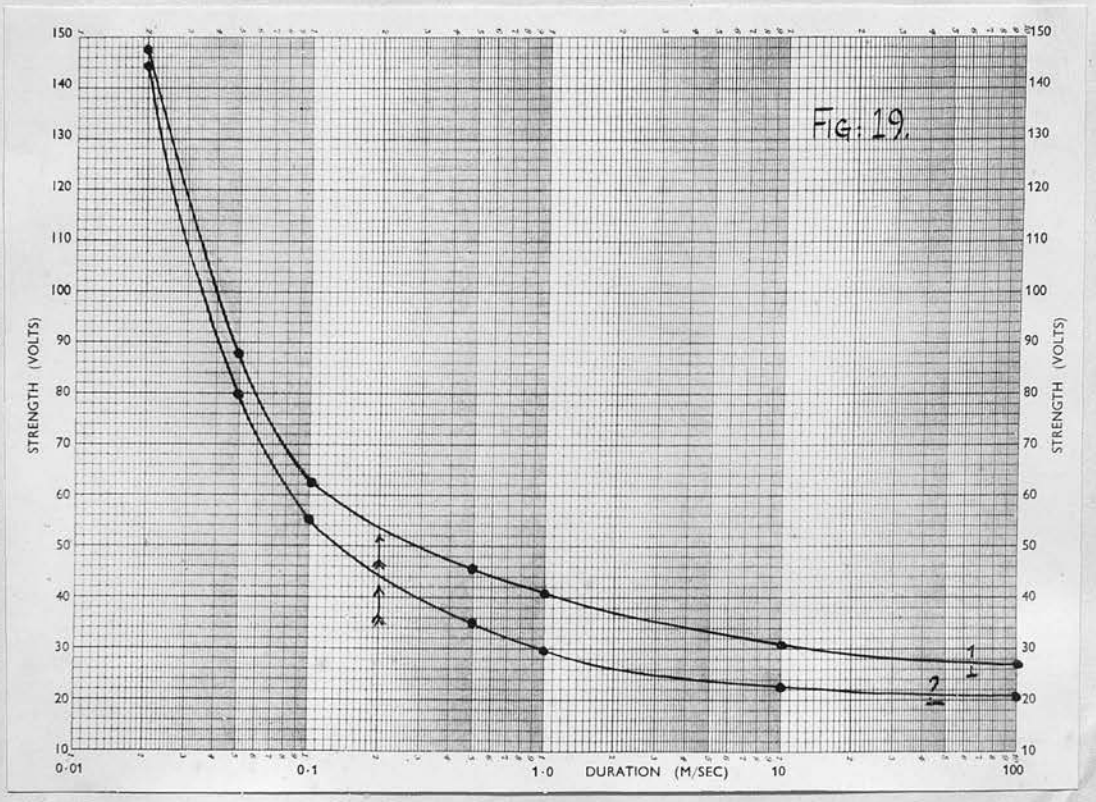
2 shows the curve 15 minutes after an intramuscular injection of 1 cc. of Pitressin (into deltoid).

The effect of venous occlusion on the S/D curve



The effect of Pitressin on the S/D curve

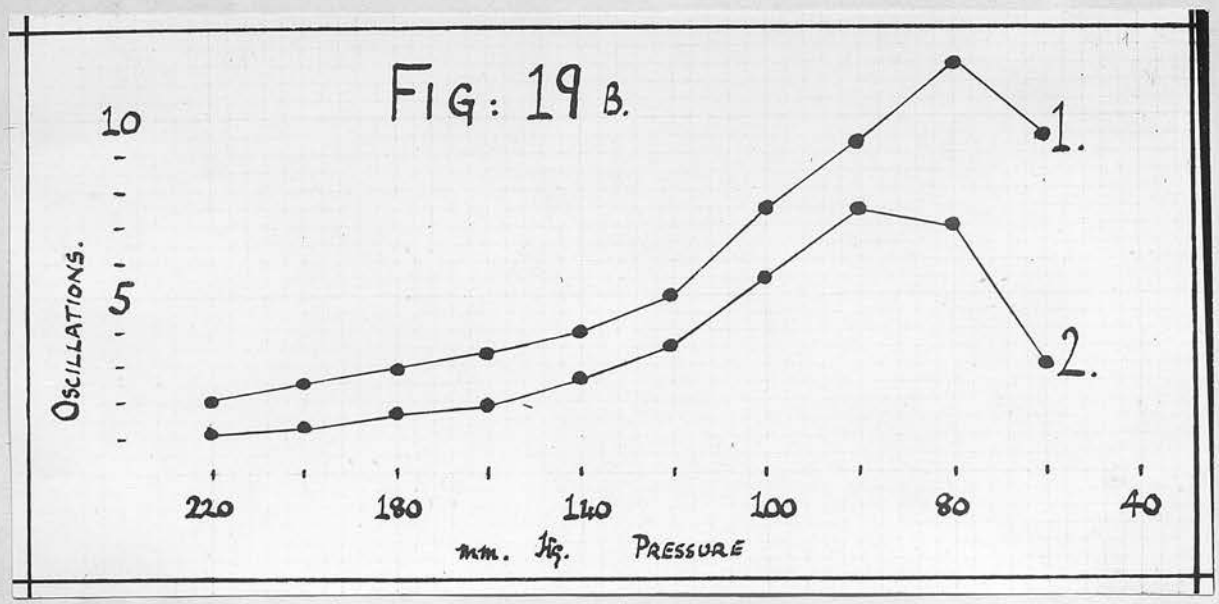
Fig. 19.



1. Curve of resting muscle.
2. 15 minutes after injection of Pitressin.

Fig. 19b.

Oscillometry of the calf before and after Pitressin



1. Oscillometry before Pitressin.
2. Oscillometry after Pitressin.

Fig. 20.

The S/D curve of denervated muscle in peripheral nerve injury

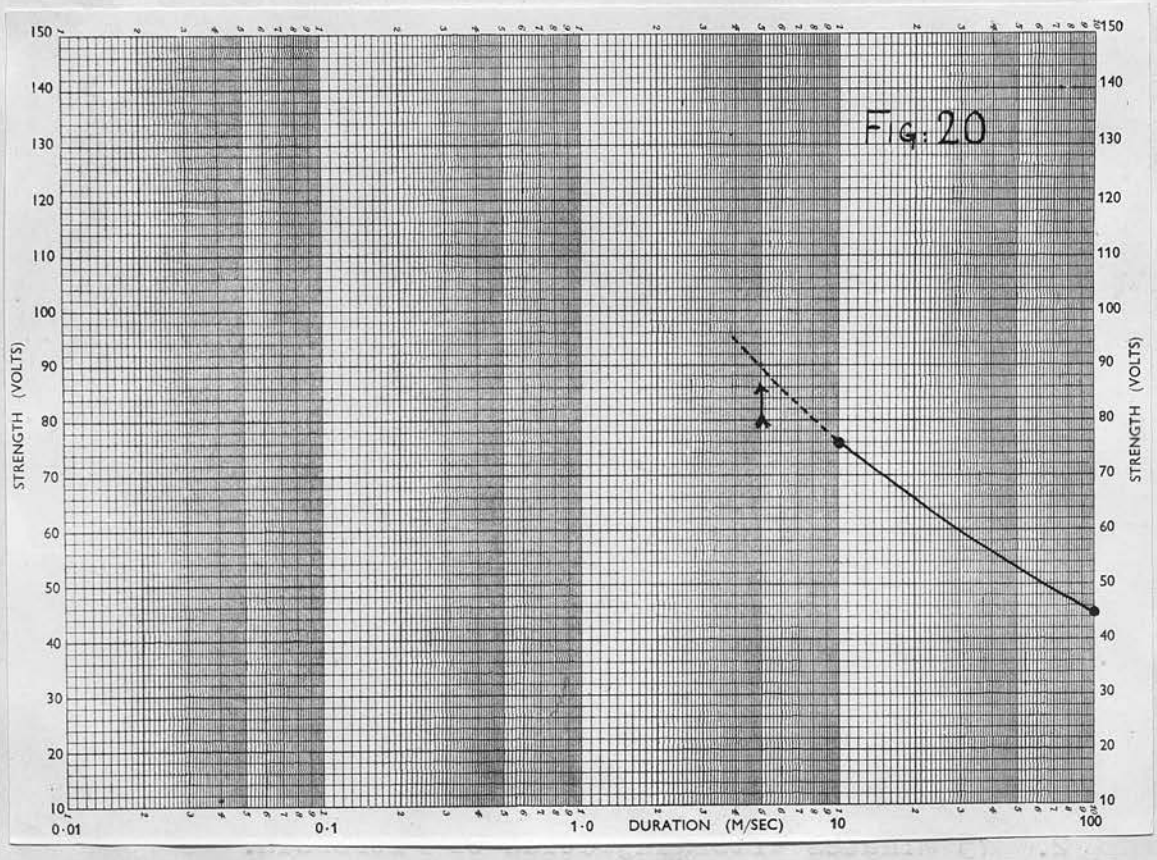


Fig.19B shows in graphic form the results of oscillometry on the contralateral calf before and after the injection, and leaves no room for doubt about the efficacy of the injection.

These figures illustrate the fact that there is a variation in the appearance of the strength-duration curve of normal muscle under different environmental conditions. The general shape of the curve seems to be altered but little, but the height may vary about 10 volts either side of the mean normal line. These findings are in agreement with those of Ritchie (1944(a) - see Fig.49,(p.56).

Obviously, slight variations in the curve from the normal must be interpreted with great care; this problem is discussed in more detail later.

The strength-duration curve characteristic of denervated muscle

Fig.20 shows the curve obtained from the tibialis anterior of a case of lateral popliteal lesion - proved complete at operation - 9 weeks after the injury. There was no response to stimuli of shorter than 10 M/S duration. Sluggish contractions were obtained in response to the stimuli of 10 and 100 M/S duration; the rheobase was 45, chronaxie five, and Lassalle's index 18125.

The reaction of degeneration, as described by Erb (1868) comprised:-

1. Loss of galvanic and faradic excitability of nerve.
2. Loss of faradic excitability of muscle.
3. Persistance of galvanic excitability of muscle which may be increased.
4. The "law of contraction" is altered so that the anodal closing current becomes equal to or greater than cathodal closing contraction.
5. The contraction becomes long drawn out.

Pollock (1945) has summarised the objections to this description:

1. Percutaneous faradic stimulation of denervated muscle is effective throughout denervation in the cat although the threshold is increased.

2. Responses are obtained in man when exposed denervated muscle is stimulated, and in some cases percutaneous stimulation is effective although the voltage required for this is very high and not normally bearable.

3. There is doubt as to whether there is an increase in galvanic excitability.

4. There is also doubt as to whether polar reversal is an infallible sign.

Pollock (1945), a result of his observations on experimentally produced sciatic nerve lesions in the cat, concluded that faradic stimulation was effective throughout denervation, the threshold rising till about the 30th day and then remaining constant. He found that the threshold current for galvanic stimulation fell slightly following denervation and remained at this low level until about the time of return of voluntary power when there was a rise again.

There is no fundamental qualitative difference between galvanism and faradism, the essential difference being the duration of the effective stimulus. Using the Ritchie stimulator, long duration stimuli may be regarded as being comparable to galvanism and short duration stimuli to faradism.

We have found that there is a loss of response to stimuli of short duration in most cases, but it has been noted (Bowden R.E.M. - unpublished work) in ulnar nerve lesions - where the muscle examined, 1st dorsal interosseous, is separated from the exploring electrode by only a very thin layer of tissue - that response to short duration stimuli at high voltages may be obtained throughout the period of denervation.

We have been unable to confirm the fall in threshold for long duration stimuli. It is clear, however, from the curves obtained from denervated muscle - Fig.20 (p.32) - that the threshold at 100 M/S is not the true rheobase, and it may be that a stimulator including a stimulus of longer duration than 100 M/S would demonstrate this fall of rheobase.

The curve of denervated muscle given by the machine we have been using, is one in which there is a response to 10 and 100 M/S duration stimuli only, where the rheobase is rather higher than normal, and where the chronaxie is very much higher than normal, and between three and eight M/S.

We have found that the curve obtained from a muscle on the brink of recovery is very similar to that of a denervated muscle. Indeed, we have not been able to distinguish the two. Electromyographic examination of subsequent repeated estimation of the strength-duration curve enables one to differentiate paralysed muscle from muscle in which recovery is about to occur, but an isolated observation is confusing (see Fig.28, and 29, p.43).

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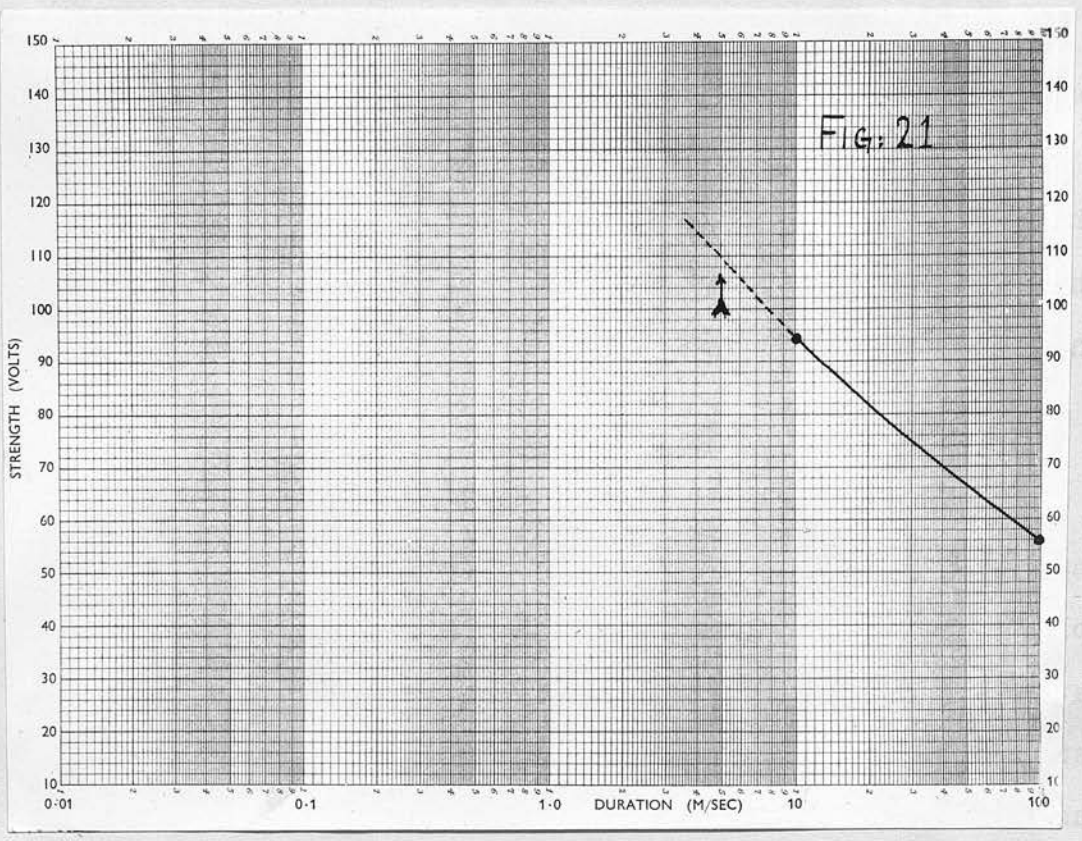
Fig. 21.

The S/D curve of paralysed muscle in Poliomyelitis



RESULTS

The curve of unaffected muscle in Poliomyelitis

Unaffected muscles in Poliomyelitis give a normal strength-duration curve (see Fig.13,p.28). This has already been discussed.

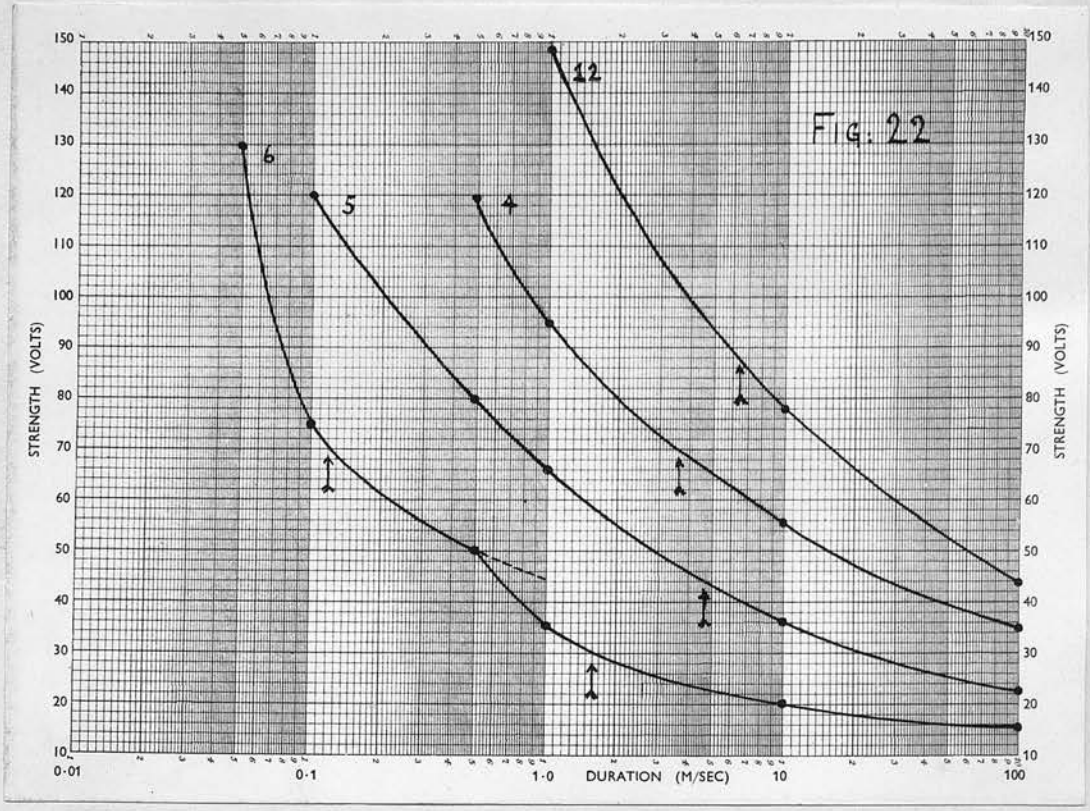
The curve of paralysed muscle in Poliomyelitis

Fig.21 shows the curve obtained from the right peronei of a severe case of Poliomyelitis with paralysis affecting both lower limbs. The muscle was wasted and showed no voluntary power; there was no response to stimulation of the lateral popliteal nerve, and there was no response in the muscle to stimuli shorter than 10 M/S. Sluggish contractions were obtained in response to stimuli of 100 and 10 M/S duration. The "rheobase" is 56, the chronaxie five and Lassalle's index 15600.

This curve is representative of a number of similar curves obtained from muscles which subsequent events showed to be completely paralysed. An electromyograph of the muscle from which Fig.21 is taken showed fibrillation only.

It is clear that the threshold at 100 M/S in Fig.21 is not the true rheobase as the curve is still sloping quite steeply in this region. This means that the actual figures obtained for chronaxie and Lassalle's

The S/D curve of partially paralysed muscle in Poliomyelitis



NOTE:

1. The curve at the 4th week with characteristics near that of paralysed than normal muscle.

2. Apparent improvement at the 5th and 6th weeks; apparent regression to a curve very like that of denervated muscle at the 12th week.

Fig. 23.

Week by week changes in the S/D curve of partially paralysed muscle in Poliomyelitis

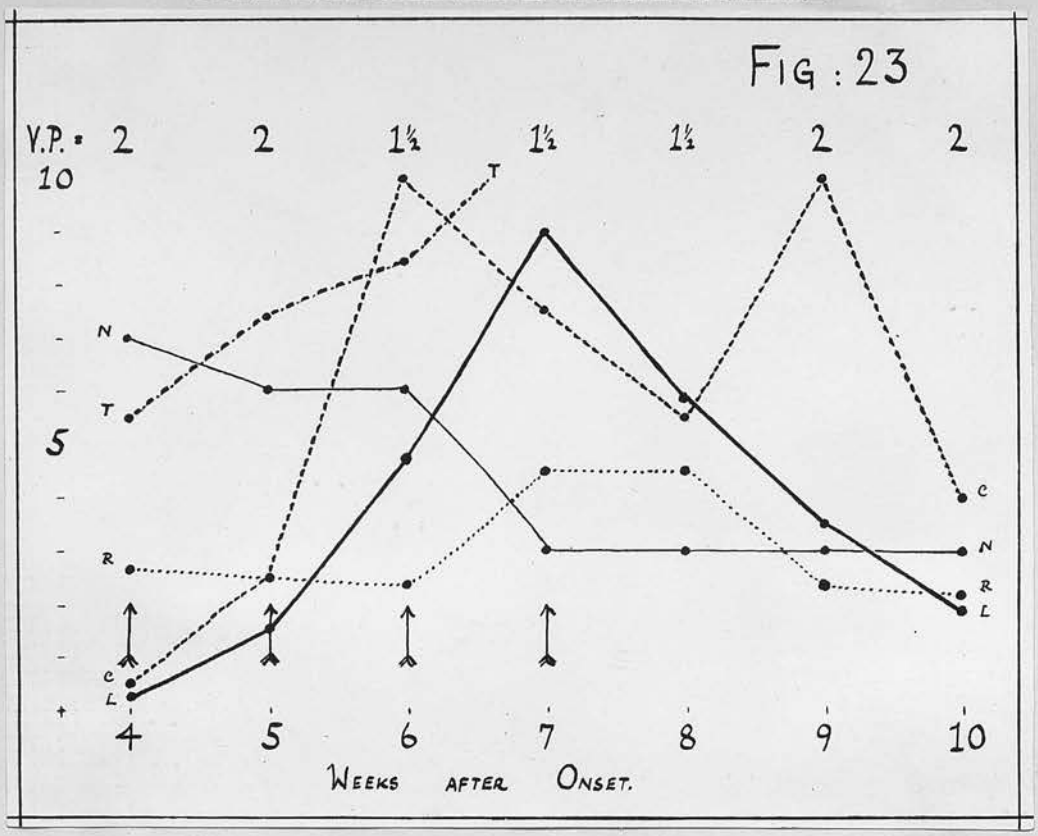
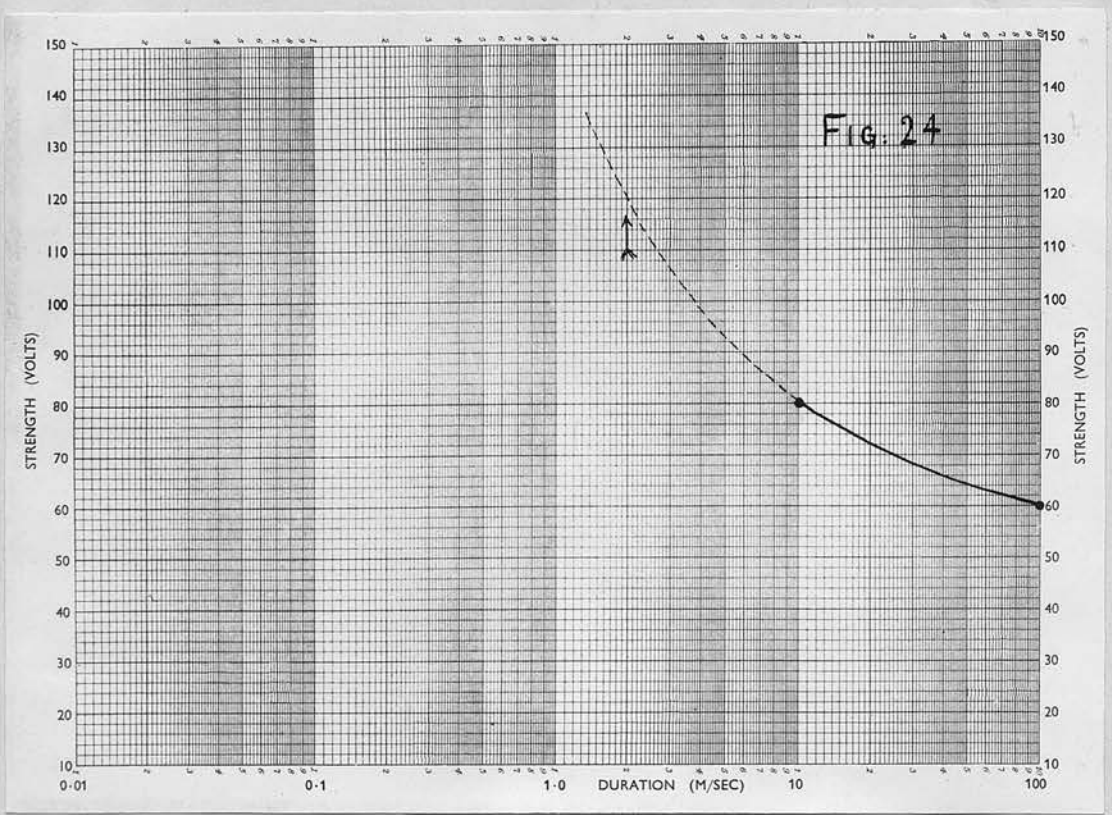


Fig. 24.

The S/D curve in a case 4 days after the onset of paralysis



R.D. is almost fully developed.

index are not strictly accurate, and not strictly comparable with figures taken from more normal curves; but since this inaccuracy occurs in all curves of this type, the figures obtained from them may be compared with those from other similar curves and with those from more normal curves provided it is realised that such figures should not be taken as absolute values.

The curve of partially paralysed muscle in Poliomyelitis

Fig.22 shows the various types of curve that are obtained from a partially paralysed muscle. The muscle examined, the right tibialis anteriorly was acting feebly (2-3½) and the curves shown are those at the 4th, 5th, 6th and 12th weeks after onset. It will be noticed that the curves for the 4th, 5th and 6th weeks descend towards the normal and that for the 12th week reverts to a type much nearer that of denervated muscle; this phenomenon is more fully discussed later under the effects of exercise on the strength-duration curve.

The curves of partially paralysed muscle shown in Fig.22 are intermediate between those for normal muscle (Fig.5) and denervated muscle(Fig.21) and are representative of a number of similar curves of partially paralysed muscle from this series of cases.

Fig.23 shows the week-by-week changes that occur in the number of effective stimuli (N), threshold at 0.5 M/S (T), rheobase (R), chronaxie (C), and Lassalle's index (L) in a partially paralysed muscle. They are similar in type to changes in these indices found in muscles which do not recover, and their significance is discussed later.

The rate of development of these variations from the normal

It is generally accepted that the characteristic reaction of denervation takes between two and three weeks to become fully developed in peripheral nerve injuries. We have found in this series that there is considerable variation in the time taken in different individuals; the cause of this variation is obscure. In some cases the characteristic curve appears in two or three weeks; in other cases it is not fully developed for seven or eight weeks, and in one case it was not apparent until twelve weeks after the onset of the disease.

Working in an orthopaedic centre rather than an isolation hospital, it has not been possible to examine cases in their earliest stages; the earliest records that we have been able to obtain were those of a case 9 days after the onset of the disease and four days after the onset of the paralysis. By this

time, the paralysed muscles in this case showed a curve characteristic of denervated muscle. Fig.24 (p37) shows the curve obtained from one such muscle - the right tibialis anterior. Three other muscle groups, the quadriceps, calf and peronei were paralysed in this case. The strength-duration curves of all these muscles were similar to Fig.24 (p.37) - characteristic of denervated muscle - and subsequent events have proved the paralysis to be permanent.

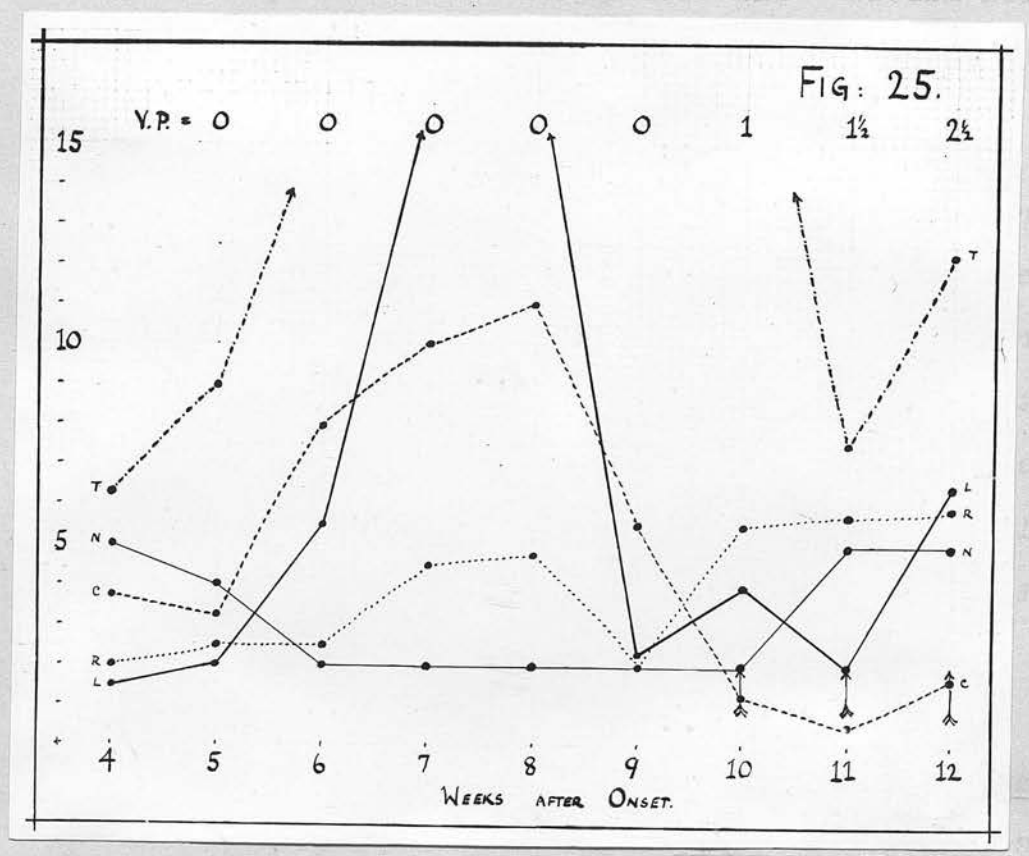
It is evident that changes in the strength-duration curve may occasionally occur very early in the disease, and it would be very interesting to record a series of curves on cases from the very outset of the disease and to see if minor variations in the curves could be found before the actual onset of clinical paralysis, and whether a prognosis of the extent of the paralysis could be based on the results of the strength-duration curve.

Changes in the strength-duration curves of paralysed muscles which recover.

Fig.25, 26 and 27 (p.40-42) show in graphic form the changes in the characteristics of the strength-duration curves of paralysed muscles that recover; these figures are representative of a number of others showing the same type of change. In one of the cases illustrated (Fig.25,p.40), the recovery was only moderate ($2\frac{1}{2}$),

Fig. 25.

Week by week changes in the S/D curve of recovering muscle in Poliomyelitis



but in the others it was complete.

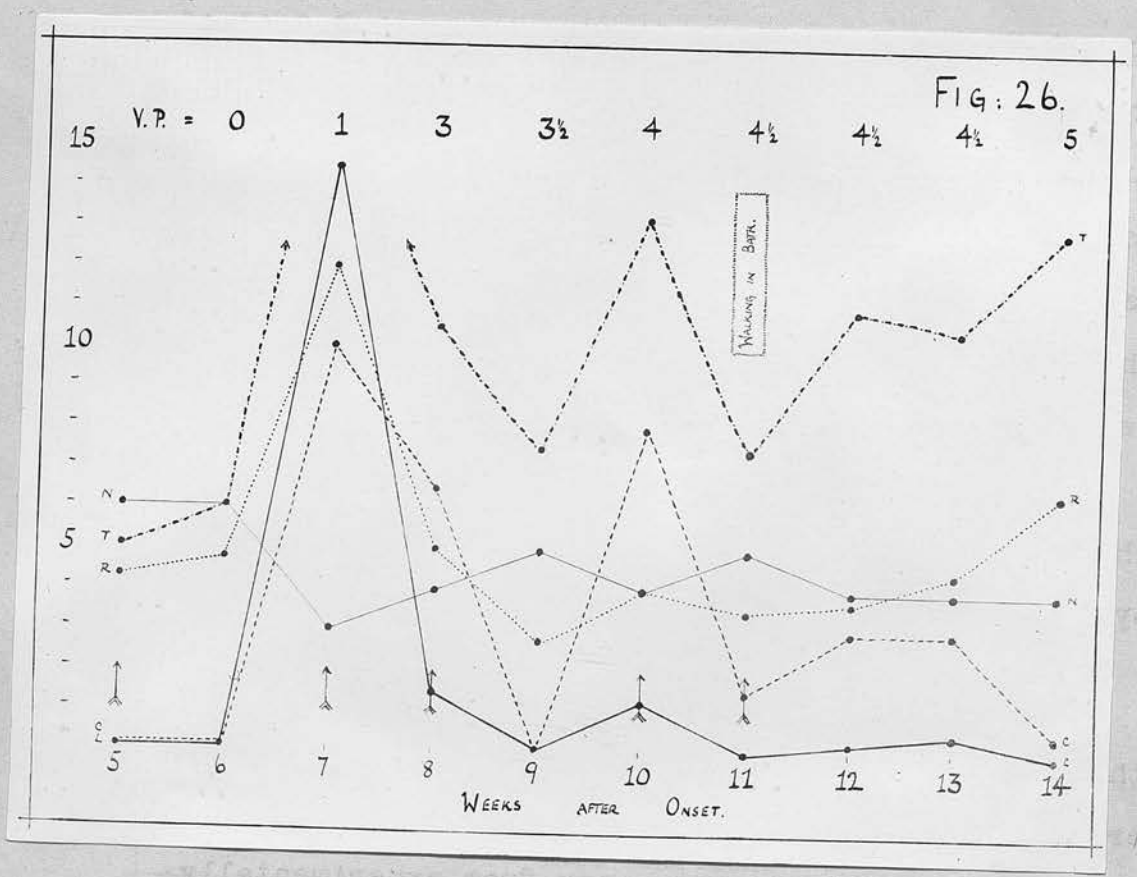
The scale and marking of these figures are all the same; the abscissae represent the number of weeks after onset, and the ordinates the value of the indices of the curve at each of these weeks. The voluntary power of the muscle is indicated along the top of the graph. The ordinates are marked 5.10.15, and these units vary for the different indices. The number of effective stimuli are represented by a thin continuous line, and the scale is 5 = 5; the rheobase is indicated by a dotted line, and the scale is 5 = 50; the threshold for stimuli of 0.5 M/S duration is represented by a dotted-and-dashed line and the scale is 5 = 50; chronaxie is represented by a dashed line and the scale is 5 = 5; Lassalles index is represented by a thick continuous line and the scale is 5 = 5000.

A glance at these figures tells one that they show the same type of change that has been described by Pollock (1945) for the curves of the gastrocnemius of cats during recovery from experimentally produced sciatic nerve lesions in the cat.

To save repetition it might be as well to say here that certain striking changes occur in the electrical reactions of muscles that recover, but that these changes may occur before, after or coincidental with the return of voluntary power, and in no

Fig. 26.

Week by week changes in the S/D curve of recovering muscle in Poliomyelitis



evidently regular manner. If these changes represent changes in the excitability of the tissues, it appears that excitability varies independently of conduction of the nerve impulse - indicated by the return of voluntary power; this might be explained on the supposition that they are two completely separate processes.

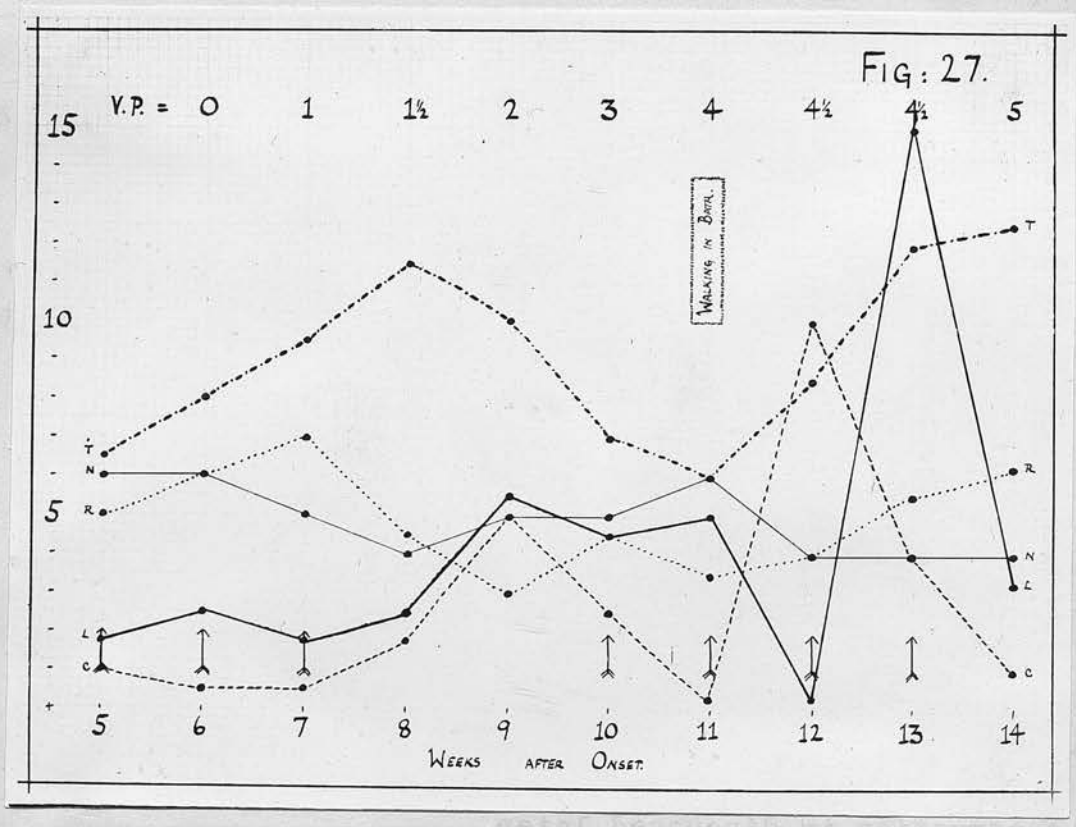
The examination of most of our cases did not begin until four weeks after the onset of the disease, and one would not therefore expect to demonstrate the early variations in the characteristics of the curve described by Pollock, but some of our cases showed a fall in these indices between the third and fourth weeks, and it may be that this represents the tail end of these changes.

Discontinuities are represented by arrows in the lower part of the graph. It will be seen that they occur both before and after the return of voluntary power in the muscle. The significance of this observation is discussed later.

The rheobase (indicated by the dotted line) shows the rise-fall-rise sequence described by Pollock. The threshold for stimuli of short duration (indicated by the line of dots and dashes) shows similar but more marked changes. There is a rise to a peak coincidental with recovery; this peak is some cases re-

Fig. 27.

Week by week changes in the S/D curve of recovering muscle in Poliomyelitis



presents an absence of excitability to these short duration stimuli; following this, there is a fall which is followed by a secondary rise which is sustained for many weeks; this sustained rise was not described by Pollock as a feature in peripheral nerve injuries.

Changes in chronaxie (indicated by a line of dashes) are striking; the indication of recovery is a rapid fall following on a peak of many times the normal value; following recovery, there is a second sharp rise (not described by Pollock) which may be larger or smaller than the original and which is followed by a gradual fall towards the normal. Lassalle's index, (indicated by the thick continuous line) shows two peaks in the same situations).

The length of the curve (indicated by the thin continuous line) decreases as recovery occurs, and then returns again towards the normal; there may be a secondary fall later in recovery, coinciding with the rise in the other indices.

As the muscle recovers, and for a varying period thereafter, the examination is somewhat painful; this is possibly associated with the rather higher voltages required to stimulate at this time, but our impression is that the complaint is usually out of proportion to the voltages used.

Thus, several striking changes occur in the

Fig. 28.

The S/D curve of impending recovery in a muscle

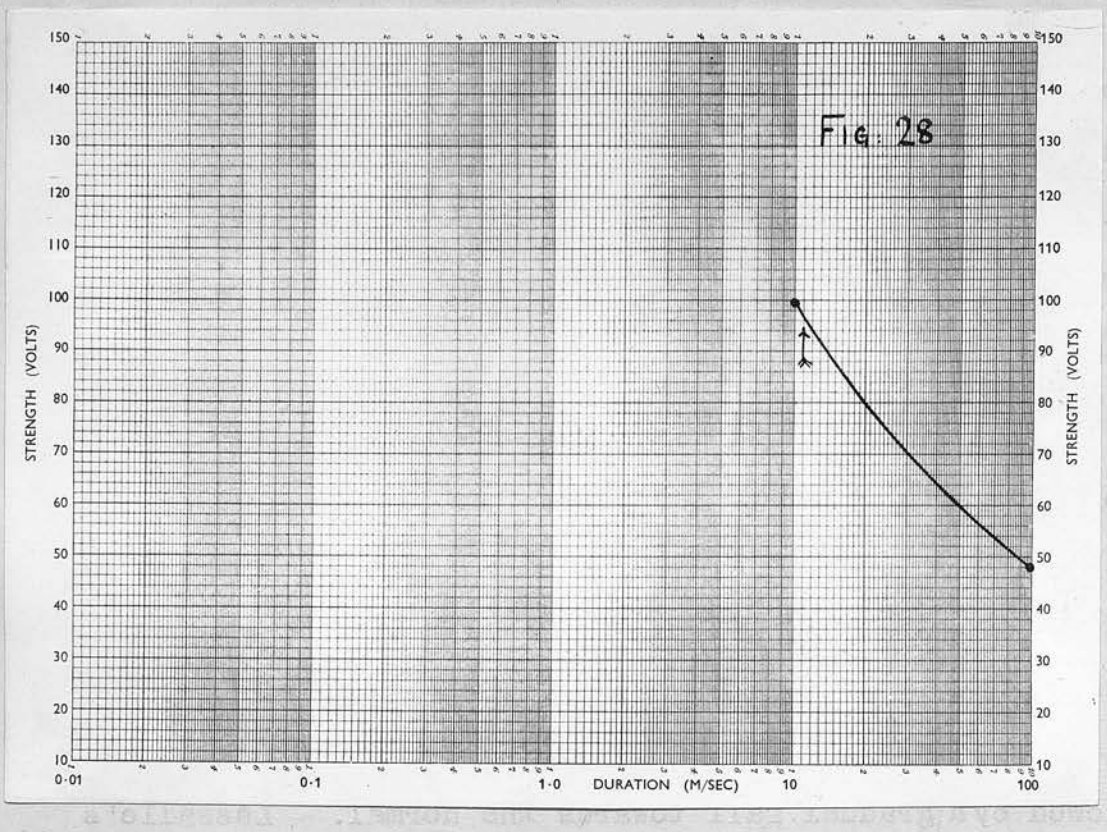
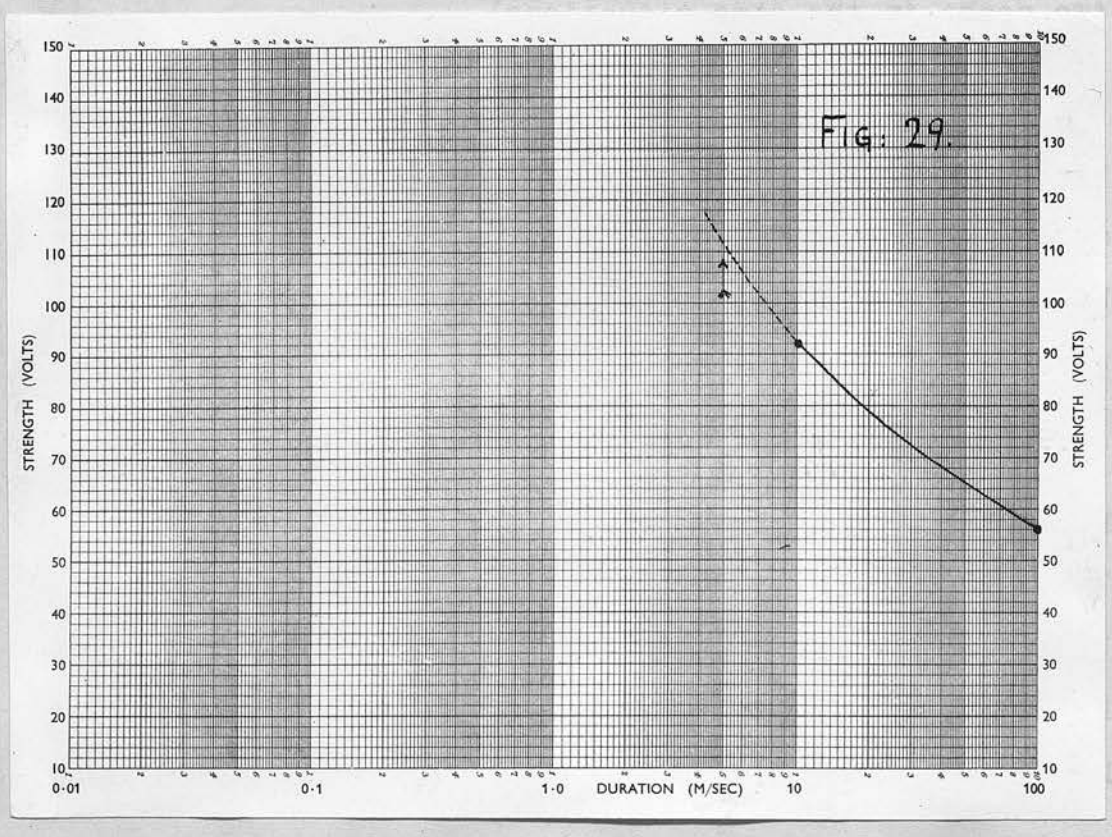


Fig. 29.

The S/D curve of a denervated muscle



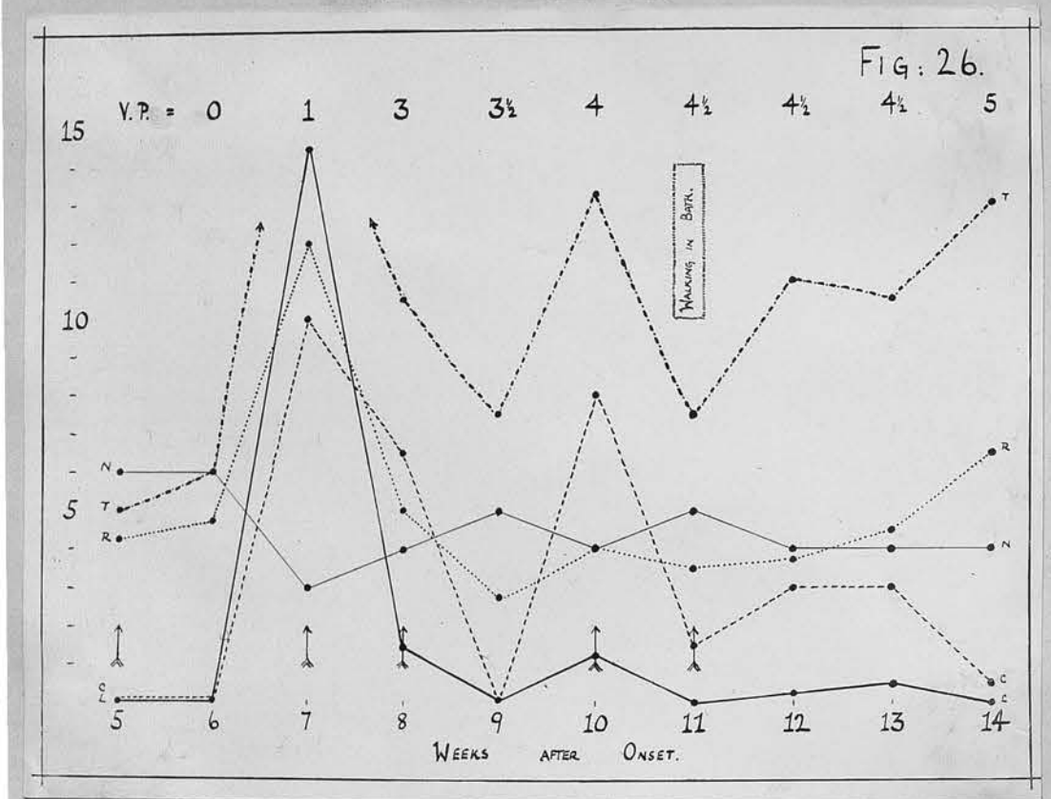
The chronaxie of recovering muscle is much higher than that of denervated muscle, i.e. the slope of the curve is steeper.

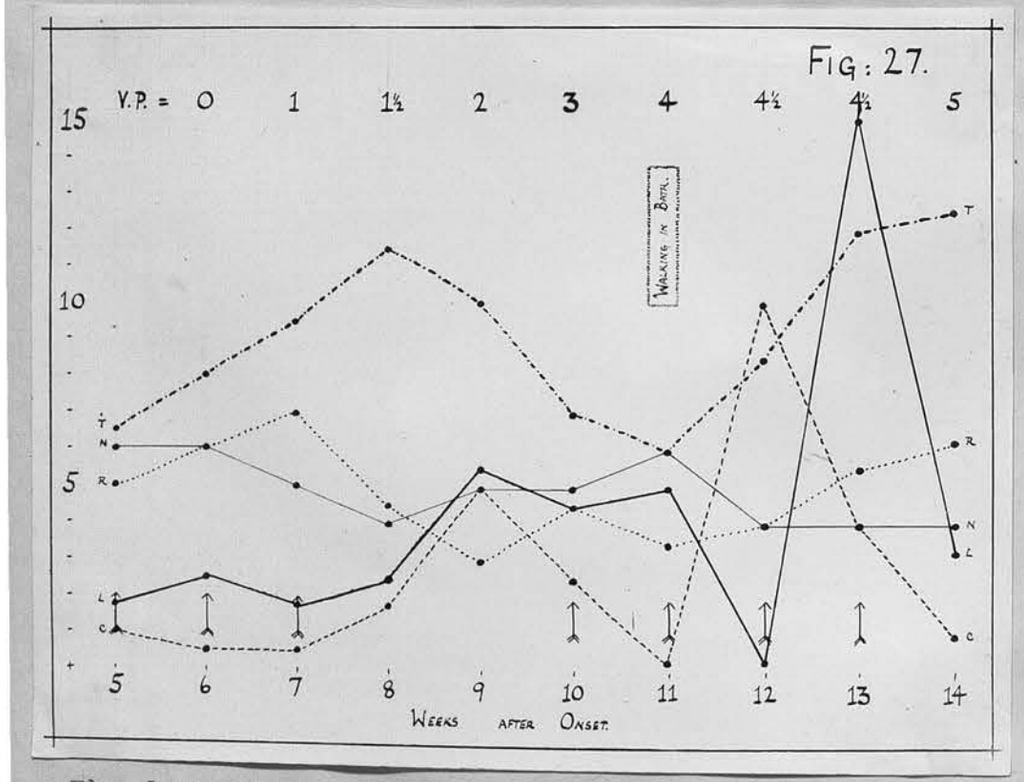
strength-duration curve as recovery proceeds. These changes may be summarised by saying that muscles that recover retain the electrical reactions characteristic of innervated muscle except around the time of return of voluntary power, when the curve is not unlike that of denervated muscle. These changes may be distinguished from superficially similar changes occurring in paralysed muscles by the fact that they are superimposed on a relatively normal baseline and, between the peaks, the indices fall to a normal level.

The curve characteristic of impending recovery.

Just as recovery occurs, the strength-duration curve obtained is not unlike that of denervated muscle. Fig.28 shows the curve from a tibialis anterior about to recover, and Fig.29 shows the curve of a denervated tibialis anterior. The curve of the denervated muscle is short, high and steep (2 effective stimuli, rheobase 56 and chronaxie five); the curve of recovering muscle is also short and high but tends to be steeper (2 effective stimuli, rheobase 48 and chronaxie 11), and it is this unusual steepness that distinguishes them. In our experience this distinction is not reliable, and we have not been able to differentiate the curves of paralysed and recovering muscle with accuracy and consistency. Electromyographic examination on subsequent estimation of the

Recovering muscle



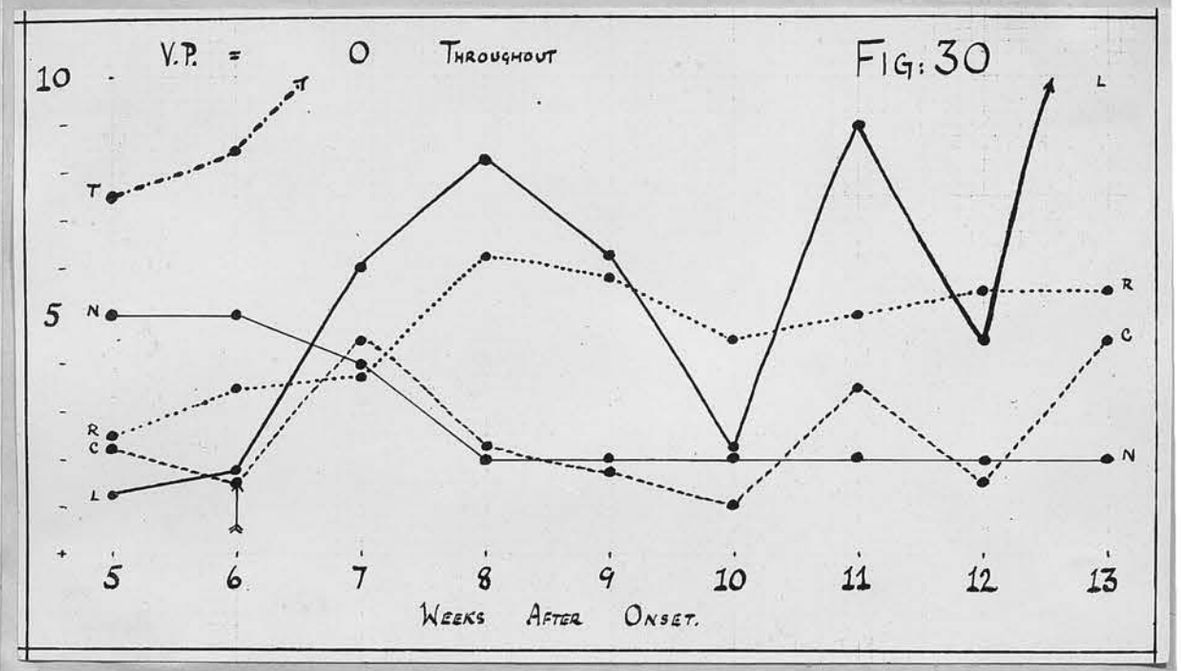


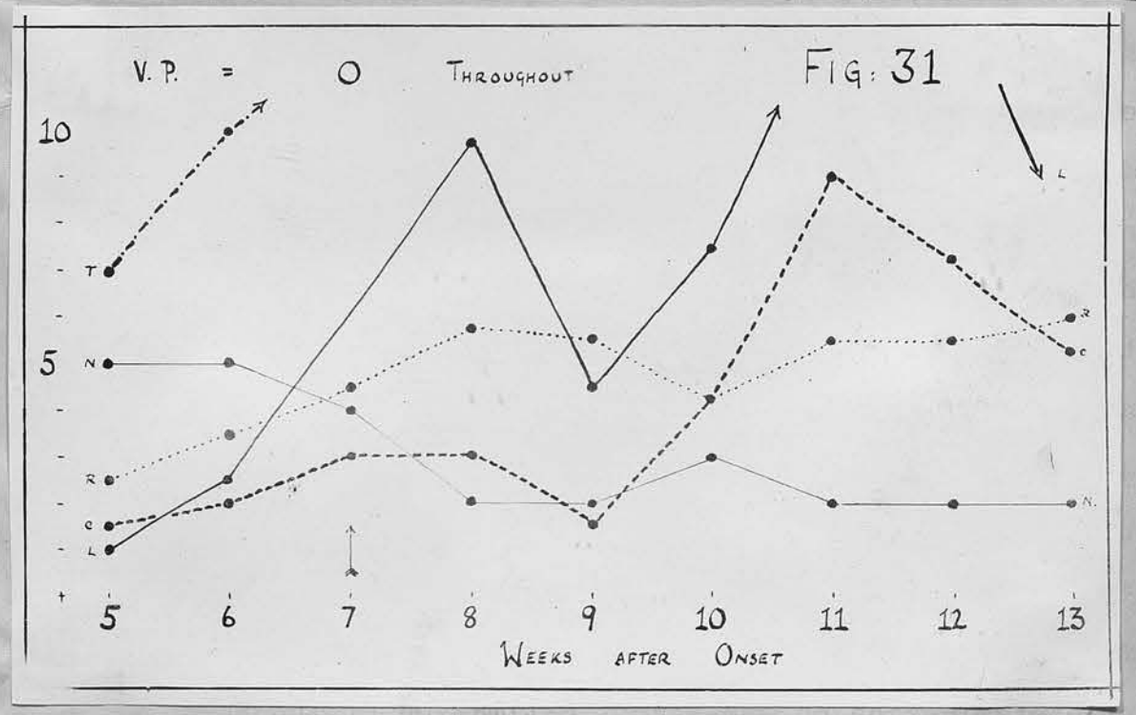
The fall of the characteristics following on the peak brings them down to a normal level.

Fig. 30 & 31.

Week by week changes in the S/D curves of paralysed muscle in Poliomyelitis

Paralysed muscle





The fall of the characteristics following on the peak does not bring them down to a normal level.

strength-duration curve does, however, enable a distinction to be made.

Changes in the S/D curves of permanently paralysed muscle

Figs.30 and 31 show in graphic form the changes that occur in the curves obtained from the right and left tibialis anterior of a case which showed no subsequent recovery in these muscles. These may be compared with Figs. 26 and 27 which are taken from the right and left tibialis anterior of a case which appeared just as severe at the outset, but in which there was a good ultimate recovery.

In the curves of the paralysed muscle, discontinuous curves were recorded at the 6th week in one case and the 7th week in the other in contrast to the persistent discontinuities in the curves of the muscle which recovered.

The rheobase shows a rise to a peak but this is not followed by a significant fall to a (temporary) normal level as in the case of recovering muscle. The threshold for short duration stimuli rises to a peak representing an absence of excitability to these stimuli, and this does not return.

The chronaxie also shows two rises as in the case of the muscles that recover, but between these rises it does not fall to a low level. The length of the

curve falls to the 2 level and remains there. Lassalle's index shows peaks similar to those of chronaxie, but again there is no marked fall to a low level between them. Denervated muscle thus shows changes in the strength-duration curves which are at first sight not unlike those shown by recovering muscle, but on closer examination it is seen that these changes are superimposed on an abnormal baseline in contrast to the relatively normal one shown by recovering muscle.

Changes in the S/D curves of partially paralysed muscles

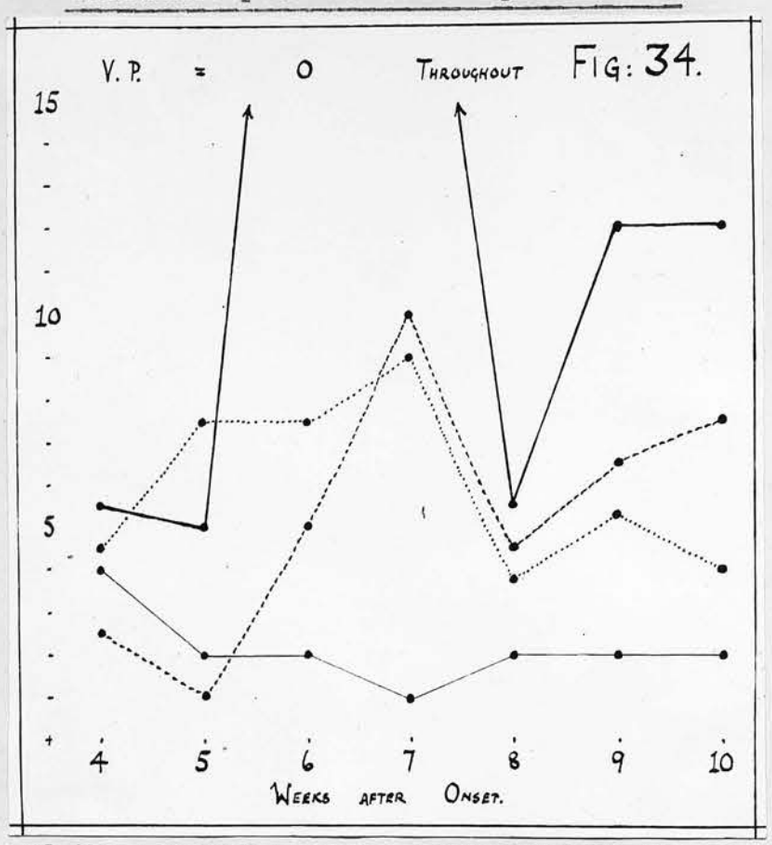
Partially paralysed muscles show the same type of changes as denervated muscle; these are illustrated in Fig.23. Perhaps a proviso should be inserted in this statement. We have not been able to obtain records of partially paralysed muscles in which the recovery was good; it is to be expected therefore that our records will show only the changes resulting from degeneration in a partially paralysed muscle, and it is possible that changes similar to those found in recovering muscle would be shown by other partially paralysed muscles in which there is a good recovery.

Early prognosis based on the strength-duration curve

Fig.32 shows the curves of the paralysed muscle illustrated in Fig.30; these curves are intermediate in type as late as the 11th week after onset.

g. 34 & 35.

Week by week changes in a paralysed muscle showing a high chronaxie peak not falling to normal



The fall of the characteristics following on the peak does not bring them down to a normal level.

The S/D curve at the 4th week of this muscle (Fig.34) showing a bad prognosis

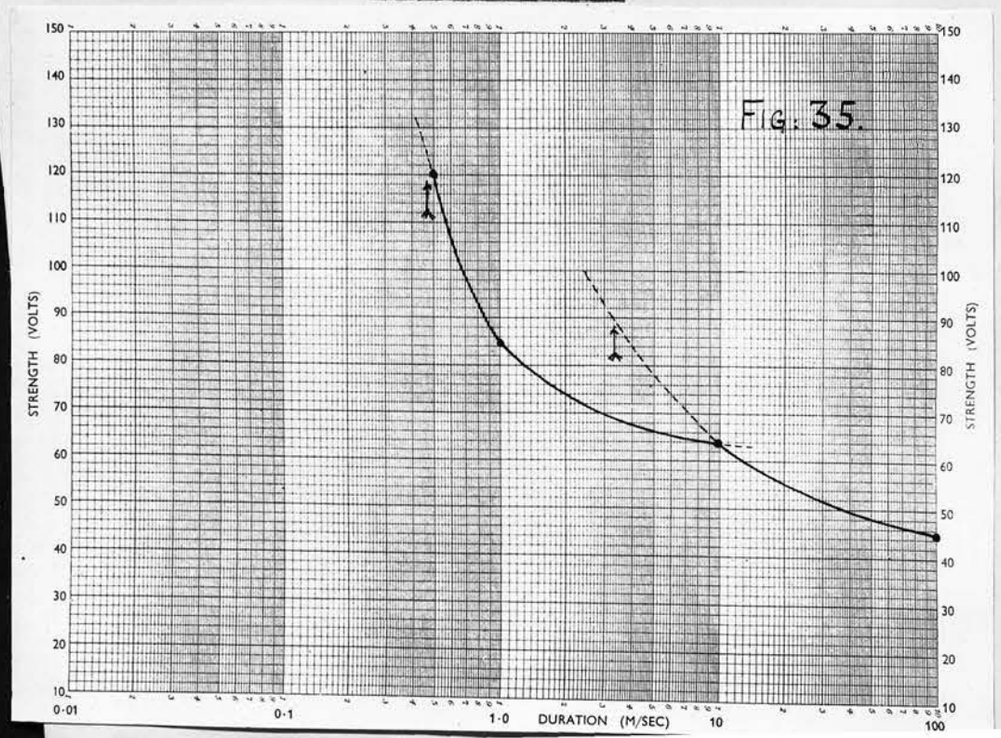
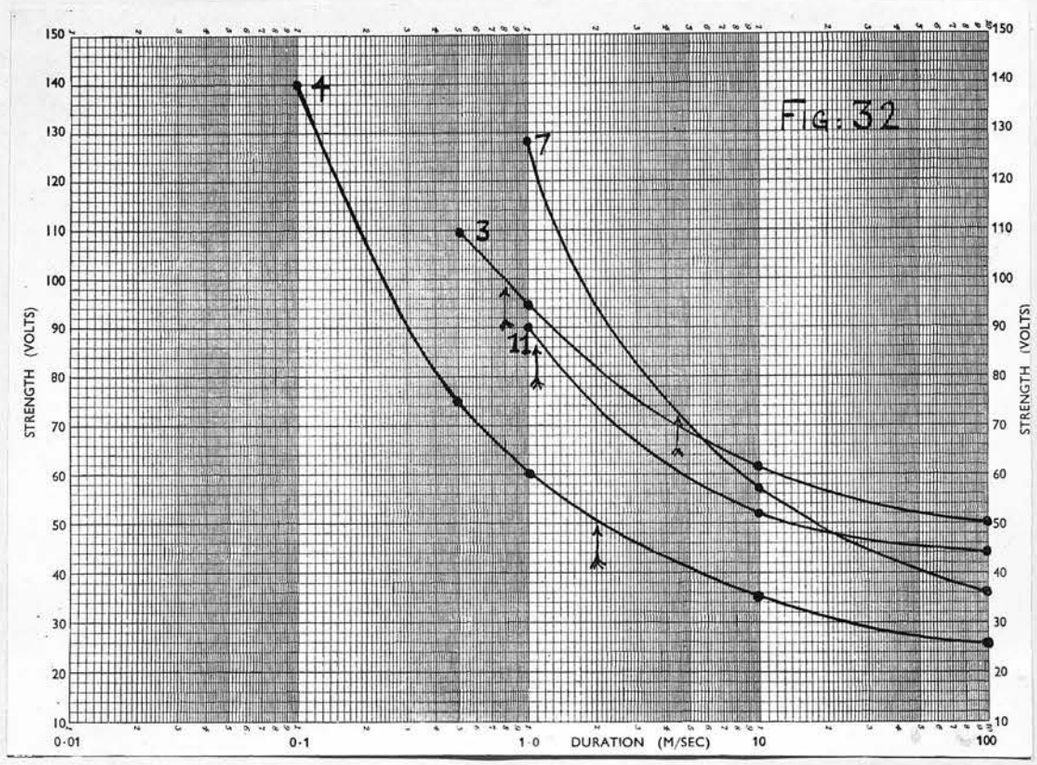
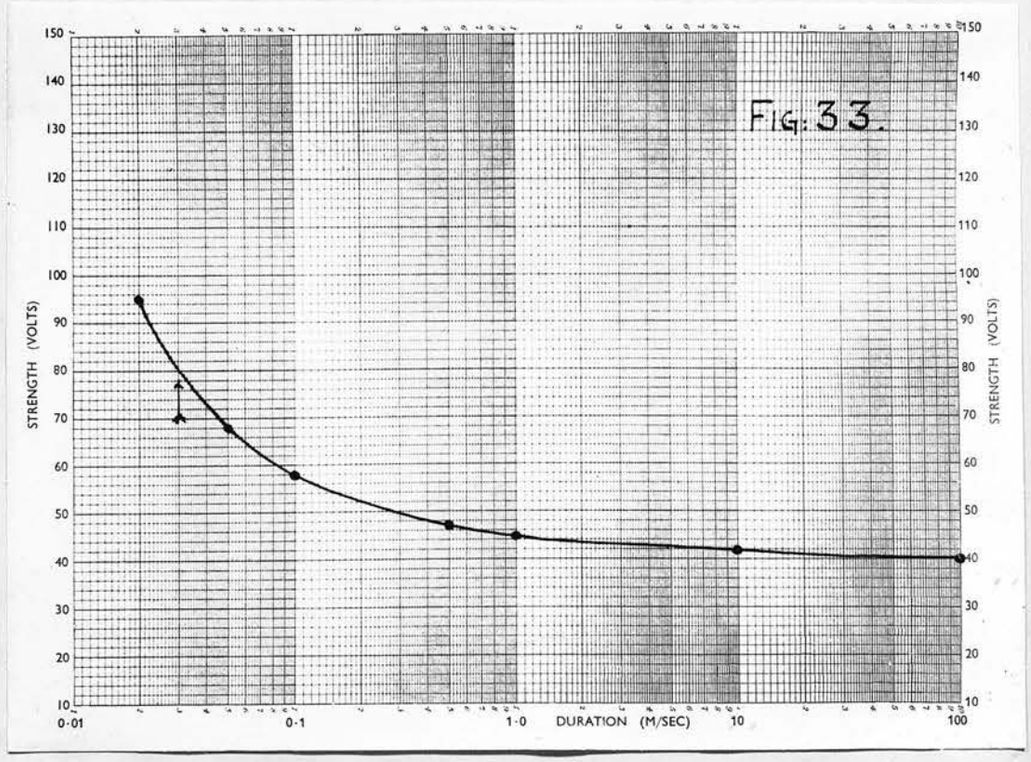


Fig.32 & 33.

The S/D curve at the 4th week showing a bad prognosis



Compare 4 with -



It is possible, however, to predict much earlier than this that recovery is unlikely to occur. Fig.33 shows the curve at the 4th week after onset of the recovering muscle illustrated in Fig.26.

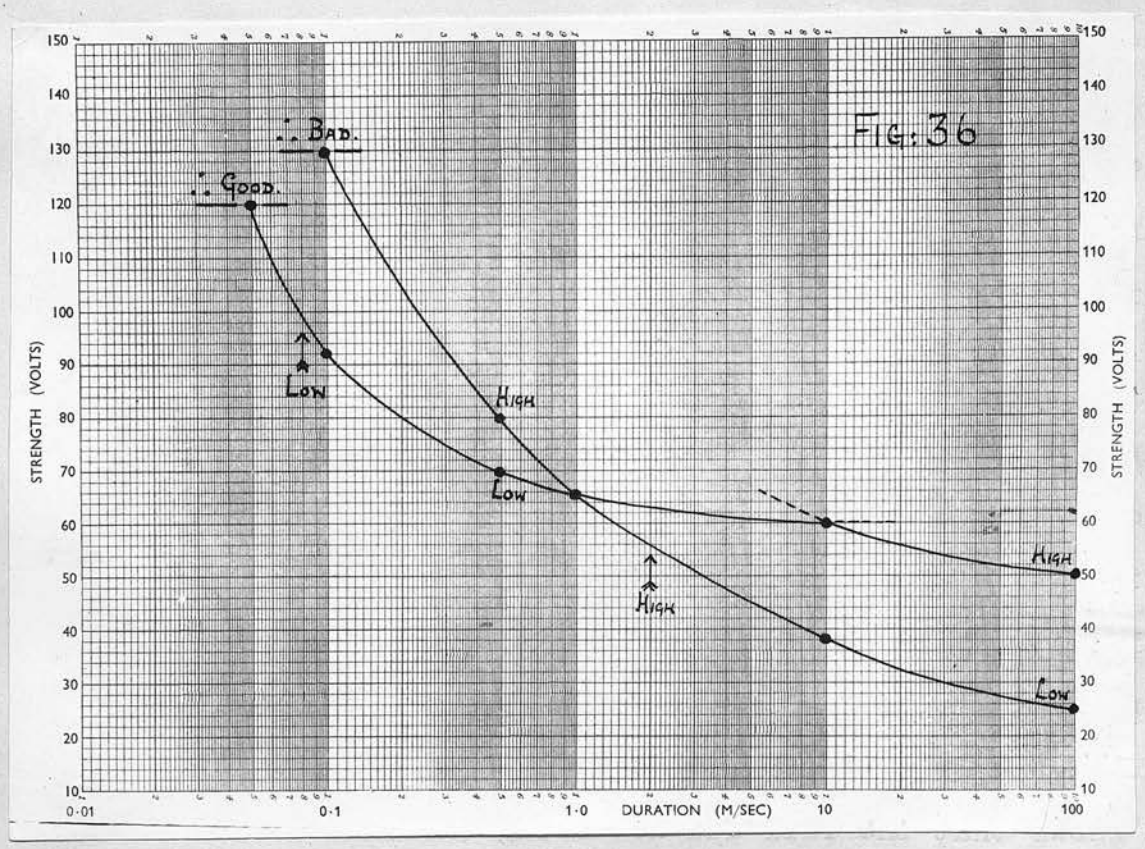
Comparing the two curves at the 4th week in the muscle that did not recover (Fig.32), the low rheobase (25), high threshold at 0.5 M/S (75), and high chronaxie (2), contrasts markedly with the high rheobase (40), low threshold at 0.5 M/S (48) and low chronaxie (0.03) of the curve of the recovering muscle (Fig.33).

The value of the characteristics of the curve at the 4th week is illustrated by Fig.34 which shows in graphic form the weekly changes in a muscle which did not recover. This shows a sharp peak and fall in rheobase, chronaxie and Lassalle's index at first sight similar to that shown in Fig.25, 26 and 27 in muscles which recovered; closer examination of Fig. 34 shows that the fall did not bring the values down to a normal level, and therefore the prognosis should be guarded. The curve of this muscle at the 4th week, Fig.35 is intermediate in type, but its characteristics are not encouraging.

The fourth week after onset is chosen as the optimum time for making a prognosis for several reasons. There is reason to believe that by then

Fig. 36.

Characteristic S/D curve at the 4th week.



degenerative changes are at a maximum (Bodian 1947). By then the early changes described By Pollock are over - although, with further experience of these changes, this in itself might not be an indication for delaying the attempt for making a prognosis. Later than the fourth week, the curves of paralysed muscle and recovering muscle are not unlike each other - although it is possible to differentiate them. Encouraging signs at the fourth week are:-

1. Five or more effective stimuli.
2. Relatively high rheobase (40).
3. Relatively low threshold at 0.5 M/S (70).
4. Relatively low chronaxie (0.5).
5. Relatively low Lassalle's index.

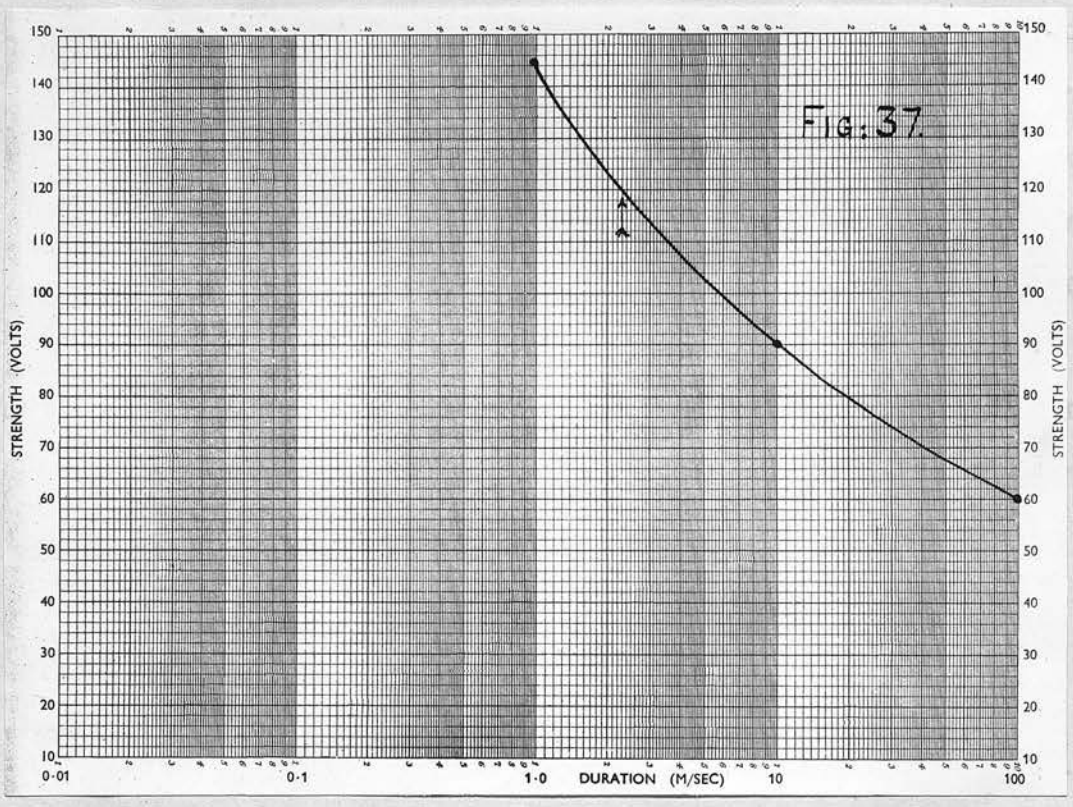
Signs on which a guarded prognosis should be made are:-

1. Four or less effective stimuli.
2. Relatively low rheobase.
3. Relatively high threshold at 0.5 M/S.
4. Relatively high chronaxie.
5. Relatively high Lassalle's index.

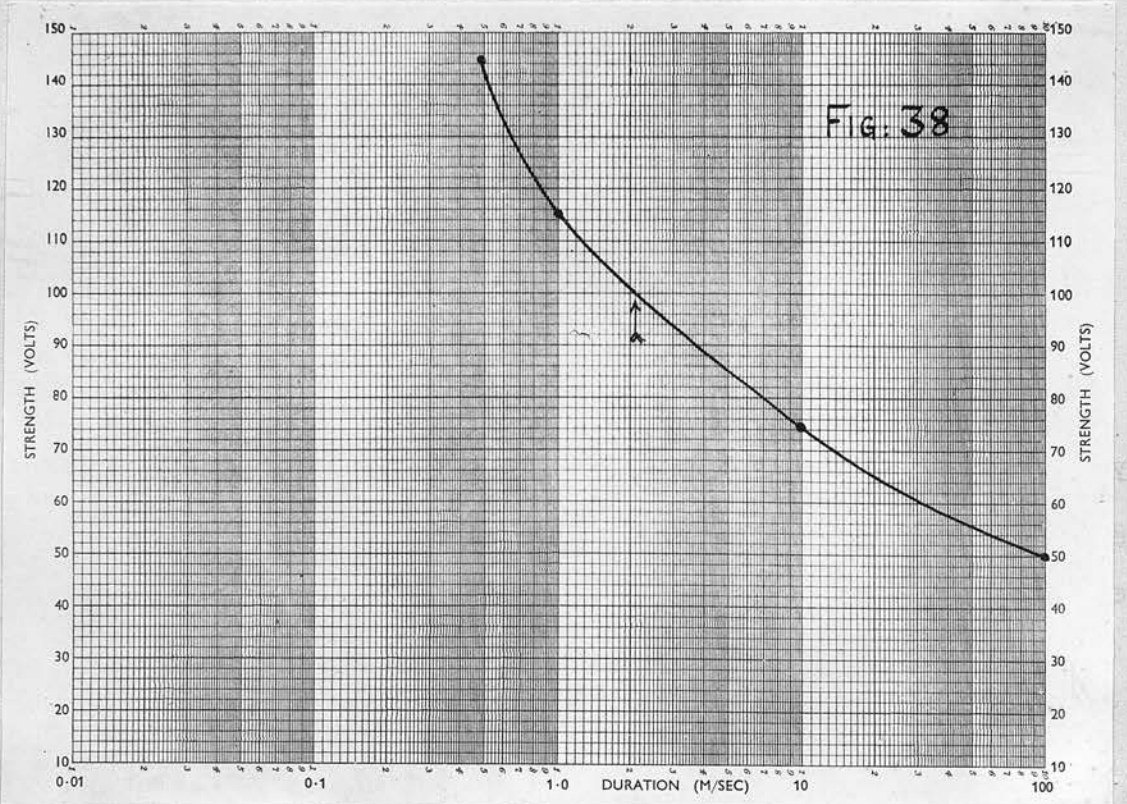
The interpretation of an isolated curve at any time must be made with extreme care; the actual figures obtained are dependent on the technique, and even minor points must be attended to with great care if the results are to be of any value.

Fig. 37 & 38.

The S/D curves illustrating prognosis at the 4th week - ba



The S/D curves illustrating prognosis at the 4th week - bad



Even greater care is needed in the interpretation of the curves obtained from partially paralysed muscle where a few normal motor units in a superficial position may give a relatively normal curve, but the above principles of prognosis at the 4th week seem to hold good for partially paralysed muscles in which the recovery is poor; we have no data on partially paralysed muscles making a good recovery.

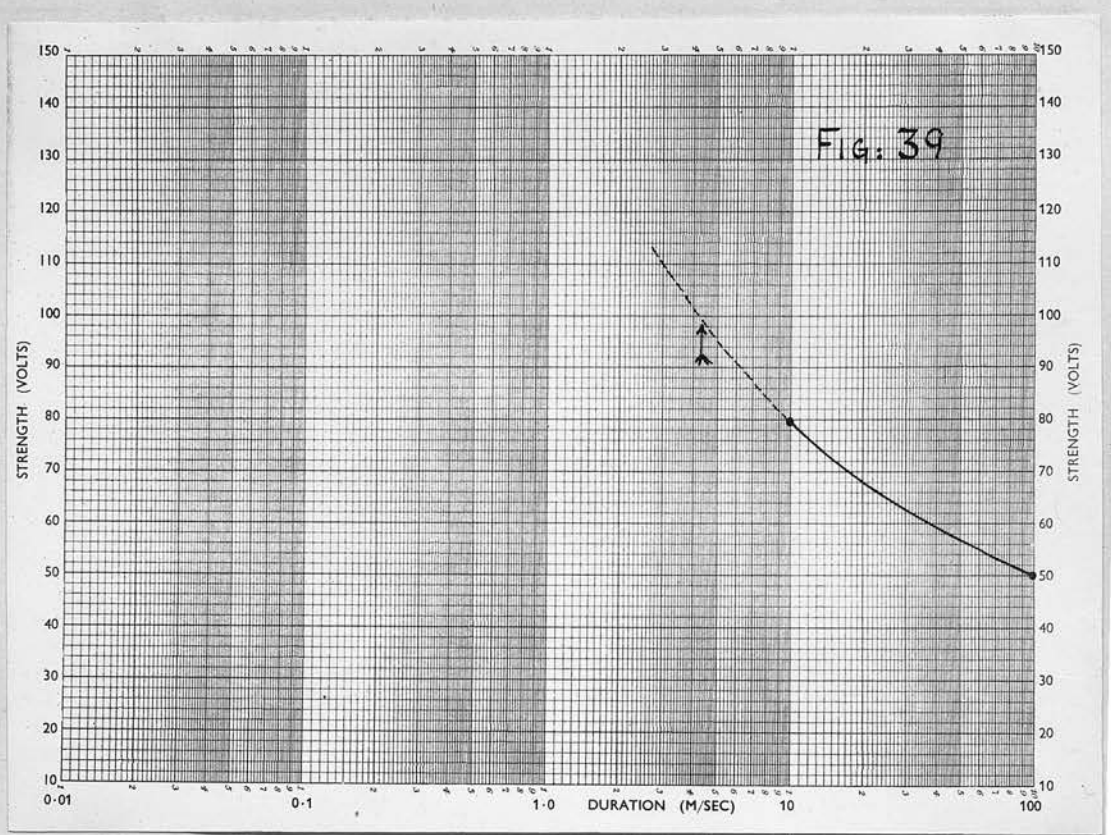
Fig.36 shows these two characteristic curves at the fourth week after onset.

Fig.37 - 45 show several examples of curves at the fourth week. Bearing in mind the five points on which the prognosis is based, it is often possible to tell at a glance into which group the curves fall. The prognosis on these figures would be:-

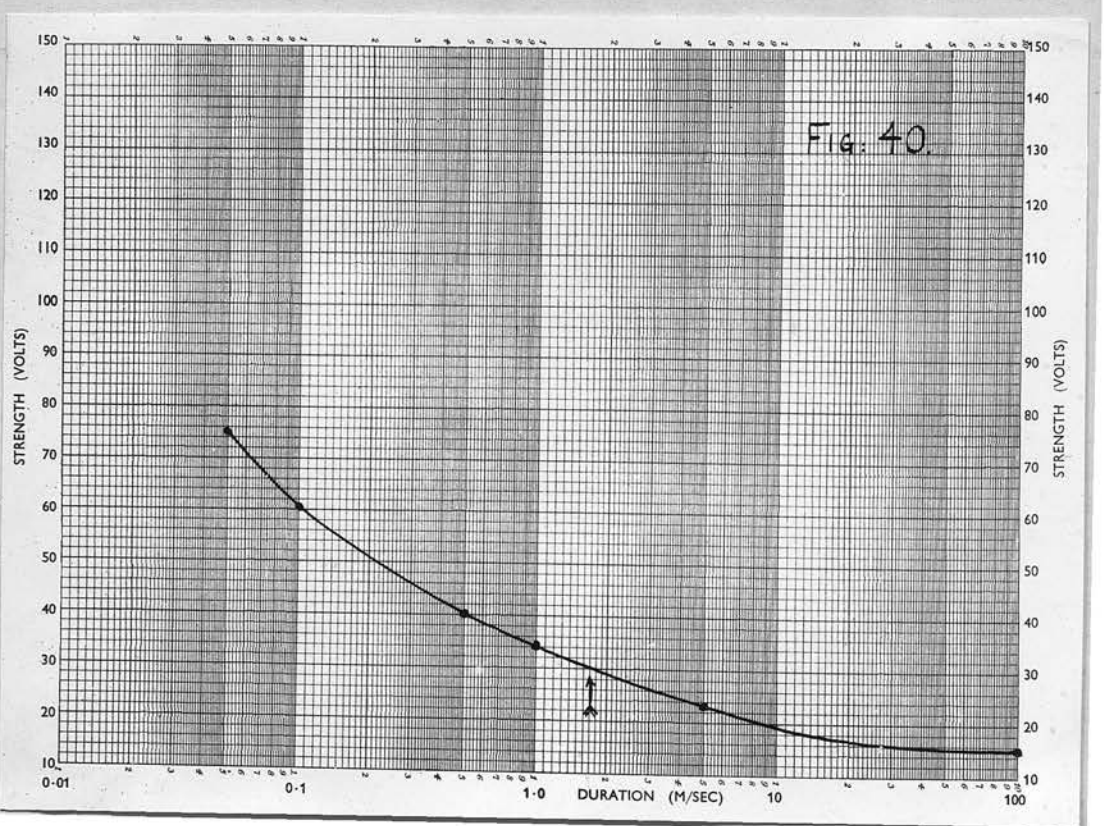
Fig.	Prognosis	Result	Muscle
37	Bad	No recovery	Biceps
38	"	"	F.D.S.
39	"	"	Triceps
40	Good	Good	Orbicularis oris
41	Encouraging	"	Peronei
42	Good	"	Tibialis anterior
43	Poor	No recovery	" "
44	"	"	Peronei
45	Bad	"	Calf

Fig. 39 & 40.

The S/D curves illustrating prognosis at the 4th week - bad



The S/D curves illustrating prognosis at the 4th week - go



Some of these curves in which the prognosis is bad appear quite encouraging at first sight, and it is evident that some more exact method of assessment than mere inspection is necessary. By a process of trial and error, we have devised an index which we have found useful for this purpose; we have not sufficient material, however, to be dogmatic about its accuracy. The index is:-

$$1 = C\,(t - r) + t.$$

Where t = threshold at 0.5 M/S

r = rheobase

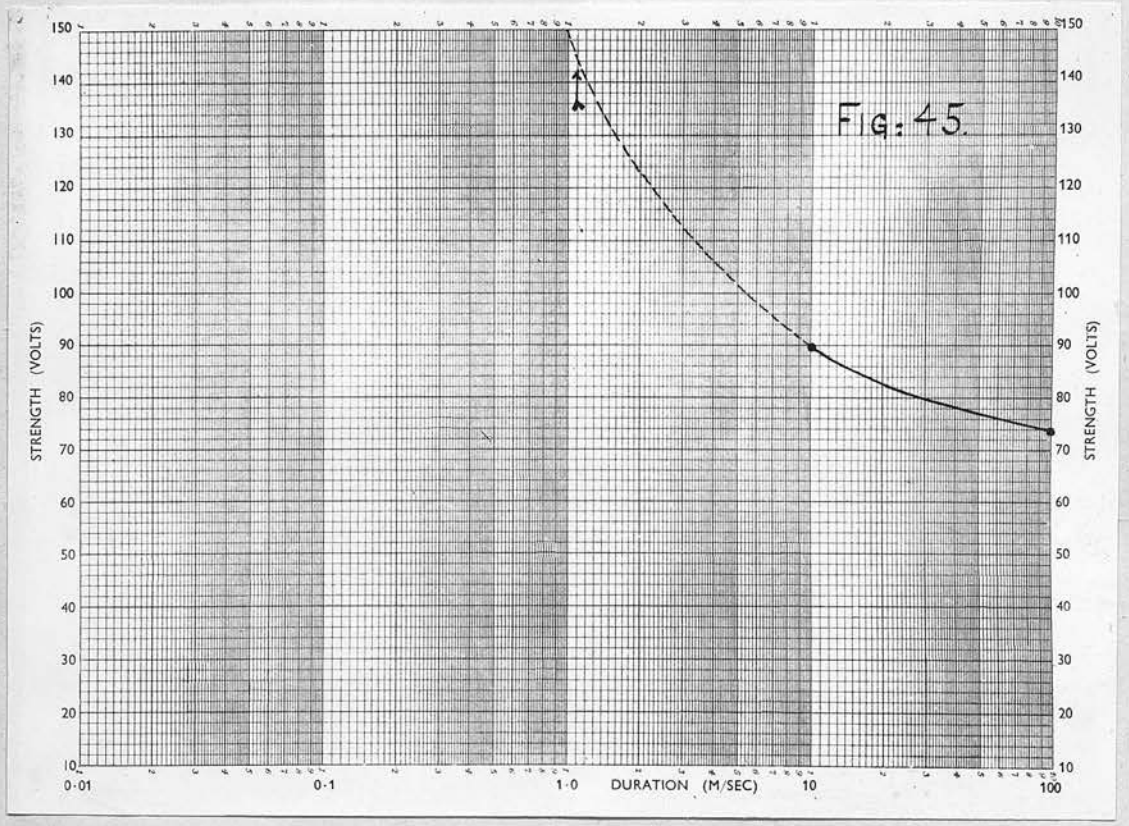
C = chronaxie

It is based on the observations that the main difference between the curves of muscles that recover well and those of muscles that do not is that in the former there is but little difference between the rheobase and the threshold at 0.5 M/S, and that the threshold at 0.5 M/S is less affected by incidental factors than the rheobase.

$(t - r)$ gives the difference between the two thresholds involved, and is really an indication of the slope of the curve; the difference in the resulting figures for recovering and paralysed muscle is exaggerated by multiplying them by the chronaxie - another indication of the slope. For discontinuous curves, we have found that the chronaxie of the curve

The S/D curves illustrating prognosis at the 4th week

- bad



as a whole - although it has no real physiological meaning - is the figure that should be taken. t is then added as an absolute value in each case.

The following examples of doubtful curves show the value of this index:-

Normal value = 50 or under.
A good prognosis is based on figures of 150 or under
A bad prognosis is based on figures of over 150.

Fig:	Index	Prognosis
32	175	Bad
35	184	Bad
41	138	Good
43	245	Bad
44	212	Bad

The effects of splinting and exercise

The policy adopted for the cases in this series was one involving a minimum of immobilisation of the limbs together with early mobilisation of the patient.

The opportunities for studying the effects of splintage were therefore limited, but in the few cases we have observed, there was no change in the strength-duration curves of affected muscles that could not be satisfactorily explained otherwise.

On the other hand, the opportunities for studying the effects of exercise were numerous. Fig.27 (p.42) shows how the changes are liable to misinterpretation; in this case, a sharp rise of rheobase and chronaxie of the muscle (tibialis anterior) occurred just after the patient had been allowed to walk in the bath, and it is tempting to try and connect these two facts as cause and effect. Examination of the corresponding muscle on the other side, Fig.26 (p.41) shows that a similar rise occurred in this muscle, but this time the week before walking was allowed, and that after walking there was no significant change in the characteristics of the curve.

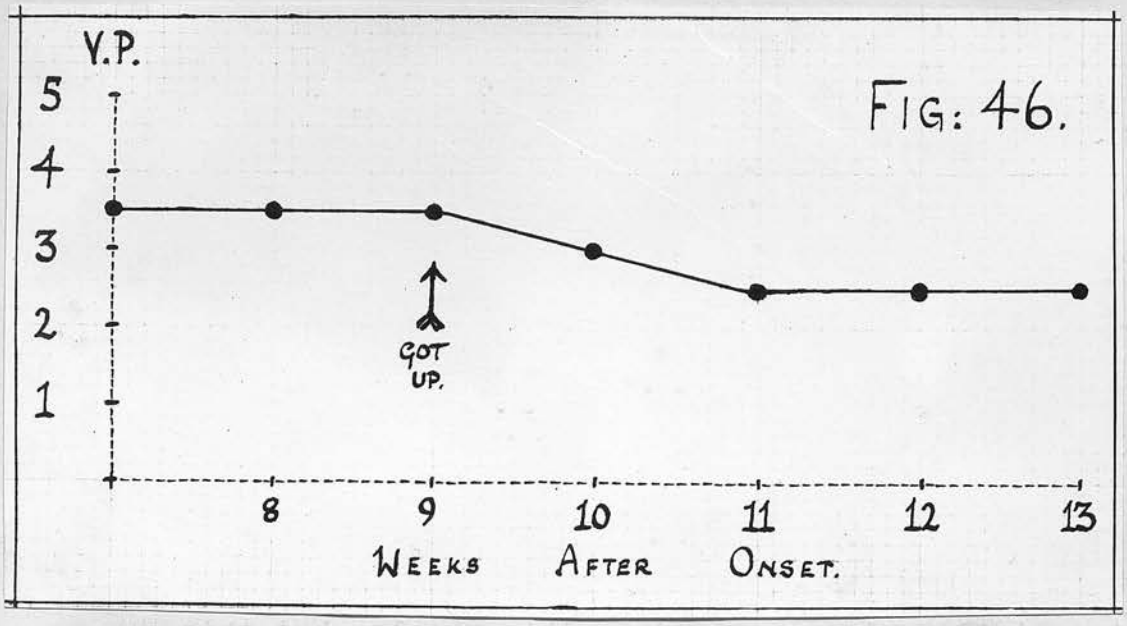
In our experience, exercise has no effect on the curves of totally paralysed muscles or of muscles which make a good recovery.

The position is not so clear in regard to partially paralysed muscles, particularly those which are just about 3 (contraction against gravity). Our clinical impression is that unless these muscles are treated with some measure of respect, they will deteriorate.

Fig.22 (p.37) shows the curves from a partially paralysed tibialis anterior. At the 4th week the curve was not encouraging and the muscle was acting feebly (2); there was some improvement in the curves.

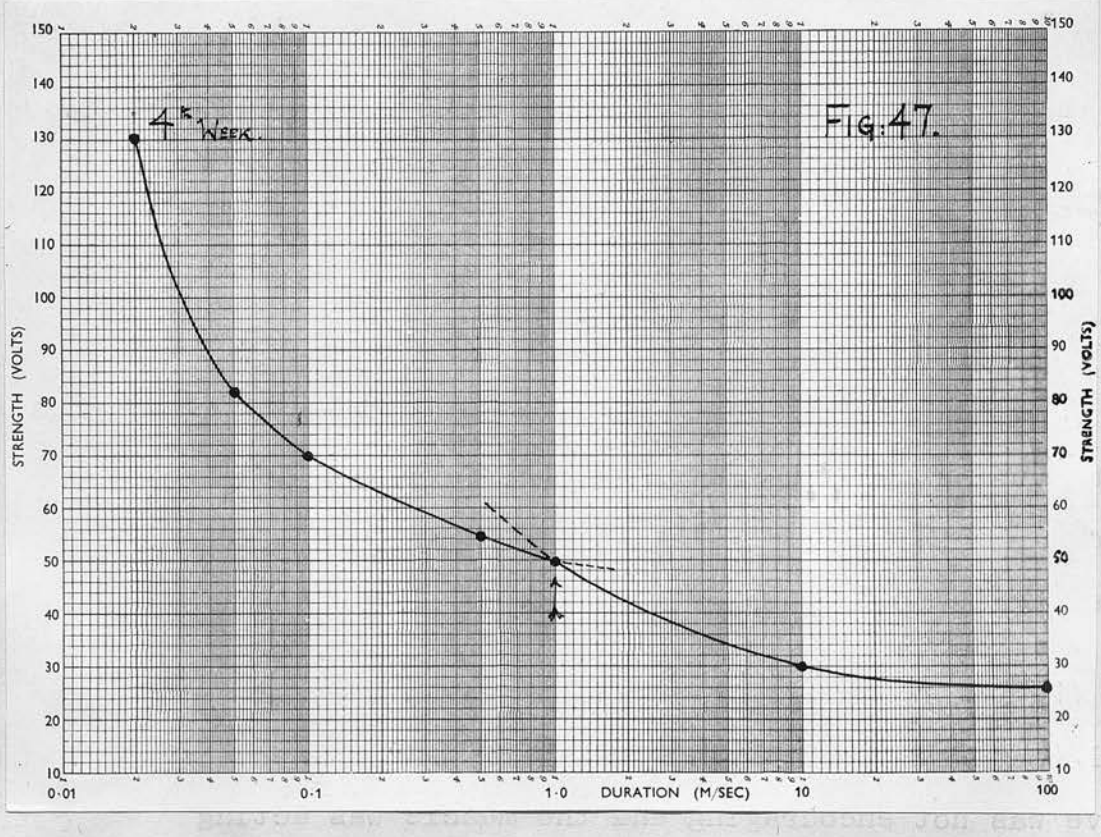
Fig. 46 & 47.

Deterioration of voluntary power coincidental with exercise



S/D curve of partially paralysed muscle at 4th week after onse

Index = 85. Prognosis good.



Walking allowed from 8th week without toe-raising spring.
Ultimate recovery poor.

and the voluntary power until at the 7th week this had risen to $3\frac{1}{2}$. At the 9th week the patient was allowed up, the curves began to deteriorate until at the 12th week there was one very like that of denervated muscle and the voluntary power fell off - Fig.46: Fig. 22.

It is again tempting to connect the exercise and falling off of voluntary power as cause and effect, but here again there is need for caution because the curve of this muscle at the 4th week was one from which a good recovery is not expected. (Index 417.)

Fig.47 however, shows the curve at the 4th week of another partially paralysed tibialis anterior and from this one would expect a good recovery; the muscle was acting feebly (2). The patient was allowed home at the 8th week without a toe-raising spring; the strength-duration curves at this time were still such that further recovery could be expected. By the 16th week, there had been no clinical deterioration, the muscle still acting at a 2, but on the other hand there had been no further improvement, and one can but wonder what would have been the effect of slightly longer rest.

The Kenny concept of Poliomyelitis

Miss Kenny's ideas on Poliomyelitis are fundamentally different from the usually accepted

views. (Pohl 1945). The most important feature is said to be "muscle spasm" which is widely present and which, if untreated, leads to permenent paralysis of the affected muscles. Muscles opposed to those in spasm are said to be "alienated" as a result of "some physiological block between them and the higher regulating centres", and these too are said to become paralysed if early steps are not taken to restore them to action. The third major feature is said to be "incoordination" which is again ascribed to a disorder of the connecting pathways between the muscles and the higher centres.

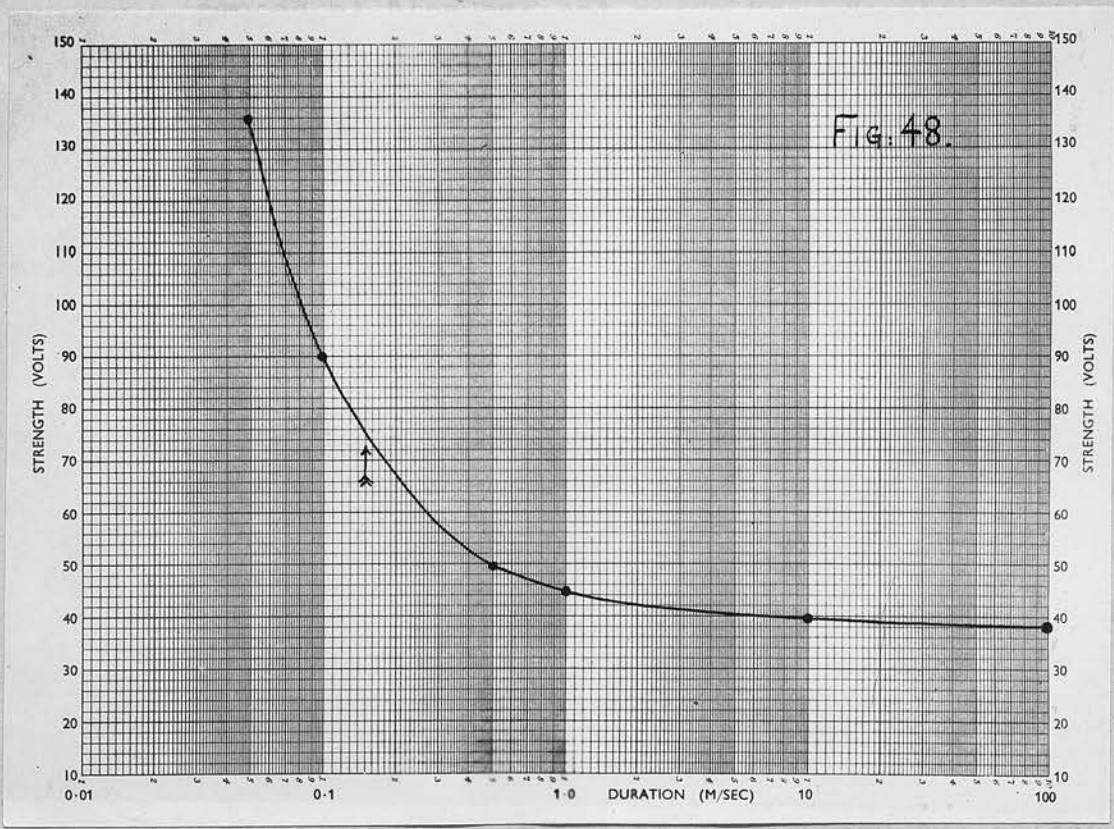
It is not proposed to discuss here the merits and shortcomings of this "new concept" of Poliomyelitis, but at all events it has served as a stimulus for further research into certain aspects of the problem, especially in America.

Since the axons supplying "alienated" muscles are presumed to be intact, the electrical reactions of such muscles should be normal. We have been unable to demonstrate normal strength-duration curves in any of the paralysed muscles in this series of cases, and conclude that "mental alienation" was not a feature in these cases.

"Spasm" in the sense of a painful limitation of joint movement was shown by a large number of

Fig. 48.

S/D curve of spastic muscle



This curve is within normal limits.

cases in this series, especially in partially paralysed muscles, but in no case was the strength-duration curve obtained from these muscles different from the curves of other muscles of comparable power not showing "spasm".

Fig.48 shows the curve obtained from the right tibialis anterior of a patient with severe spastic paraplegia. The rheobase and chronaxie of this curve are on the high side of normal, but in this case the patient was very stout, and there was slight oedema of the tissues. Other cases of spasticity gave apparently normal strength-duration curves and the conclusion is reached that this method of examination is not suitable for the detection of muscle spasm.

In answer to the question whether incoordination ever occurs, we must reserve judgement. One case, whose curves were of the intermediate variety with encouraging characteristics was quite unable to dorsiflex the foot when asked to do so, although during the determination of the strength-duration curve the dorsiflexors would snap into vigorous action. When she was asked to plantarflex the foot, however, it would be dorsiflexed after a great effort. This interesting and unusual case showed similar marked incoordination of all movements of both lower limbs and one arm.

A similar phenomenon is occasionally seen in brachial plexus lesions involving C.5 and 6 where the patient on being asked to flex the elbow brings the triceps into immediate and vigorous action and it is only after an appreciable time that there is a response in the biceps.

It may be that independent movements are acquired rather than inborn and that the knack is lost if the muscles are paralysed, and has to be relearned when recovery occurs. On the other hand, this incoordination may be a hysterical phenomenon, and in the case of Poliomyelitis described there was certainly a marked element of hysteria.

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Fig. 49.

After Ritchie. Range of variation of S/D curve

in normal muscle

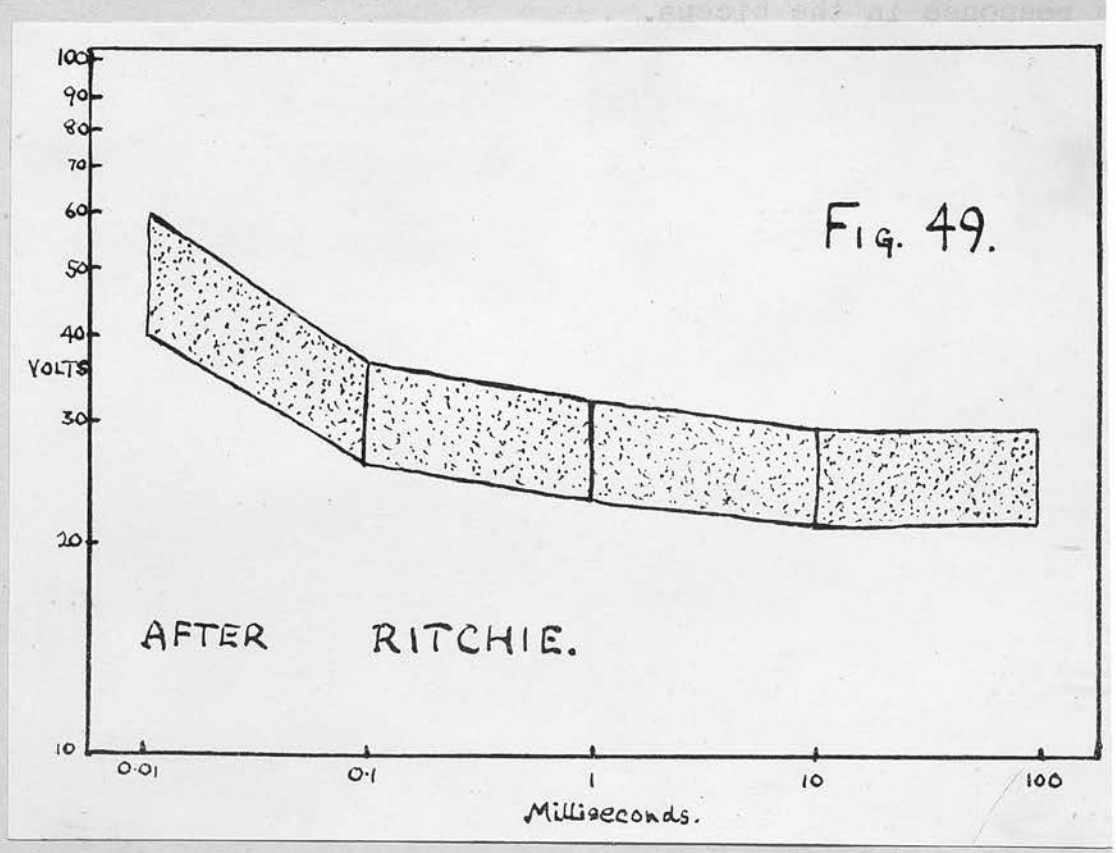


Fig.14 - 19 show variations of the same order.

DISCUSSION

The interpretation of minor variations in the curve

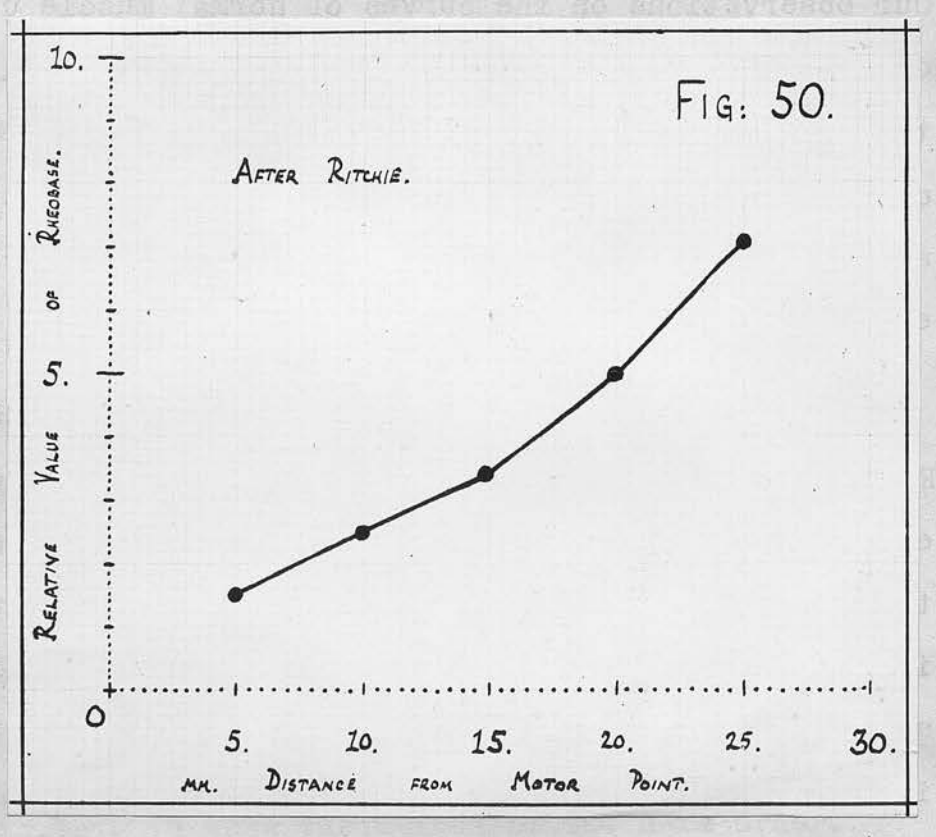
Fig.49, after Ritchie (1944), shows the range of variation that can occur in normal muscle examined under standard conditions on successive days. Our observations on the curves of normal muscle under different environmental conditions have convinced us that variations of this order do occur in the curves of normal muscle, but we have been able to obtain the same curve (within +/- 3 v.) from the same muscle examined on the same day.

In his work on peripheral nerve injuries Ritchie (1944) in considering curves recorded at different times regards as significant only those variations that fell outside the band shown in his figures; in consequence, he has not attached the same importance to discontinuities in the curves, and fluctuations in rheobase and chronaxie as has Pollock, who did not apparently follow as rigorous principles in the interpretation of his curves.

Pollock (1944,1945) is satisfied that relatively minor variations occur with consistency and in a certain sequence during denervation and recovery; and we have found somewhat similar changes occur consistently in Poliomyelitis during degeneration

Fig. 50.

After Ritchie. Variation in the rheobase with distance of the examining electrode from the motor point of the muscle



and recovery.

Fig.50, after Ritchie (1944), shows the variation in rheobase which occurs with increased distance of the stimulating electrode from the motor point of normal muscle, and this observation is used by him in support of this view that minor variations in technique are capable of producing quite marked variations in the strength-duration curve of muscle, the condition of which may in fact have remained constant throughout. We feel that while this fact can hardly be over emphasised and that although variations undoubtedly occur in normal muscle from day to day, it is still possible, if one is meticulous about minor points in technique, to attach some significance even to relatively minor variations in the characteristics of the strength-duration curve, particularly if they form part of a general trend, and that discontinuous curves are of significance.

The value of an early prognosis

If the strength-duration curve can give one an early indication of the outlook in Poliomyelitis, it may also materially affect the management of the case.

Where the electrical reactions of paralysed muscles are not encouraging, the aim should be to get

the patient up as early as possible and proceed without delay to ordering any necessary supporting apparatus; this may save the patient months of recumbency. If, on the other hand, the electrical reactions are encouraging, it is probably wiser to hasten slowly, on the assumption that there will be a gradual return of power which must not be jeopardised by throwing an excessive load on weaker muscles.

A good prognosis in a paralysed muscle raises the question of the value of electrotherapy in treatment. If it can be shown - the question will be discussed later - that some process of axonal regeneration plays a part in the recovery of Poliomyelitis, electrotherapy is a logical adjunct.

Jackson (1945) has shown that galvanism is capable of staying the process of wasting that occurs in denervated muscle and such treatment might appear desirable in Poliomyelitis for paralysed muscles exhibiting encouraging strength-duration curves. In the past, electrotherapy has indeed been extensively used.

Our feeling on the matter is that until there is more direct evidence in favour of the occurrence of axonal regeneration in Poliomyelitis, and unless one is prepared to carry out such treatment regularly and continuously for a very prolonged period, there is no indication for the use of galvanism in this disease.

If, however, one comes across a patient showing true incoordination of movement during recovery, there is a strong case to be made out for using faradism as a means of muscle re-education.

The mechanism of neuro-muscular transmission, and the significance of the variations in the S/D curves

The precise mechanism of neuro-muscular transmission is obscure; the present knowledge of the problem is given by Katz (1939), and the recent advances in this field are described by Ritchie (1945).

The axon is held to be surrounded by a selectively permeable membrane with the result that the whole becomes polarised by reason of a differential collection of ions on the two sides of this membrane. If the membrane (nerve) is stimulated locally, it is caused to "leak" with the result that there is a passage of ions across it, a flow of current, and a neutralisation of charges. This process acts on the adjacent membrane so that a wave of membrane instability, accompanied by a neutralisation of charges, passes along the axon (in both directions); this is the nerve impulse.

When the impulse reaches the end-organ, acetyl choline is released; intraarterial injection of this substance causes a short tetanic contraction of the muscle, but its intimate mode of action is not known.

It is possible that this or some other chemical mechanism is interposed between the nerve impulse and the initiation of another wave of electric excitation in muscle. There is considerable controversy as to whether this does in fact occur, particularly in the central nervous system where there is a very short period of delay at the synapse, and it has been suggested that transmission at the synapse is electrical rather than chemical.

It has now been shown that acetyl choline is a constituent of the axon itself, that the enzyme (cholinesterase) responsible for its breakdown is found in the axon sheath as well as at the synapse (it is thought that its easy recognition at the synapse is explained by the arborisation which occurs in this region giving a greater surface area of axon sheath) and that acetyl choline can be both synthesised and broken down in the time that is available during the delay at the synapse.

Thus, the main objections to the chemical theory have been answered, and, at the best, the evidence in favour of the electrical theory is indirect and contraversal.

If the nature of these mechanisms is in doubt, it is not surprising that the exact significance of the changes in the strength-duration curve accompanying recovery should be obscure. Theoretically, the curve may

represent the response of nerve, muscle or myoneural junction, and variations in any or all of these may affect the curve. It is perhaps not without significance that a muscle on the point of recovery is electrically inexcitable and electromyographically silent.

We have seen that the return of voluntary power does not regularly coincide with any characteristic electrical change in Poliomyelitis, and it is possible that conduction and excitability are two separate processes.

Discontinuities in the strength-duration curve

Discontinuities in the strength-duration curve after peripheral nerve lesions have been described by Adrian (1916), Bauwens (1943) and Pollock (1944). Ritchie did not demonstrate them to his own satisfaction but he used a machine with only five durations of stimulus and we have found that by using a machine giving seven different durations it is possible to show discontinuities that would not be apparent with five durations only. This point is illustrated by Fig.51.

Adrian (1916) showed in a case of facial palsy that when a nerve degenerates the chronaxie falls from 0.24 M/S in the normal muscle to 10 M/S in the denervated state. In studying the way in which the normal curve is transformed into the curve characteristic of denervated

muscle, he demonstrated complex curves of the types shown in Fig.6 and 7; he also demonstrated complex curves in cases of Poliomyelitis with incomplete paralysis.

Keith Lucas (1906) using fluid electrodes, examined the sartorius of the frog and found that the non-neural region gave a simple curve with a chronaxie of 3 M/S; he called this the alpha curve, and regarded it as characteristic of muscle. The neural region gave complex curves, double or even triple in form; one of these curves was similar to the alpha curve; another, the gamma curve, was similar to that obtained from the nerve trunk; the other, the beta curve had the shortest chronaxie of the three.

These observations led Adrian to conclude that during the transition from one type of curve to another, there is no gradual merging process but that both curves are present together, and that the transition consists of one curve becoming more prominent to the ultimate exclusion of the other.

He explained this phenomenon by saying that when a nerve is intact, a liminal stimulus to the muscle takes effect on the nerve endings - the more excitable tissue - alone; as degeneration proceeds, stronger and stronger stimuli are required to excite these nerve endings, and then the muscle fibres themselves respond to

stimuli of low strength and long duration. Eventually, the rapid mechanism becomes inexcitable, and the excitability of the muscle fibres alone is represented in the curve.

Lapicque, using small electrodes, failed to demonstrate discontinuities in skeletal muscle. He believed them to be artefacts, and held that the lengthening of chronaxie that follows nerve section was due to a change in the chronaxie of the muscle itself. He believed that neuromuscular transmission was only possible when the chronaxie of nerve and muscle were similar and that the failure of neuromuscular transmission following nerve section was due to an alteration of the chronaxie of the muscle.

Jinnaka and Azuma (1922) and Davis (1922) showed that the actual figure obtained for chronaxie varied with the size of the electrodes used.

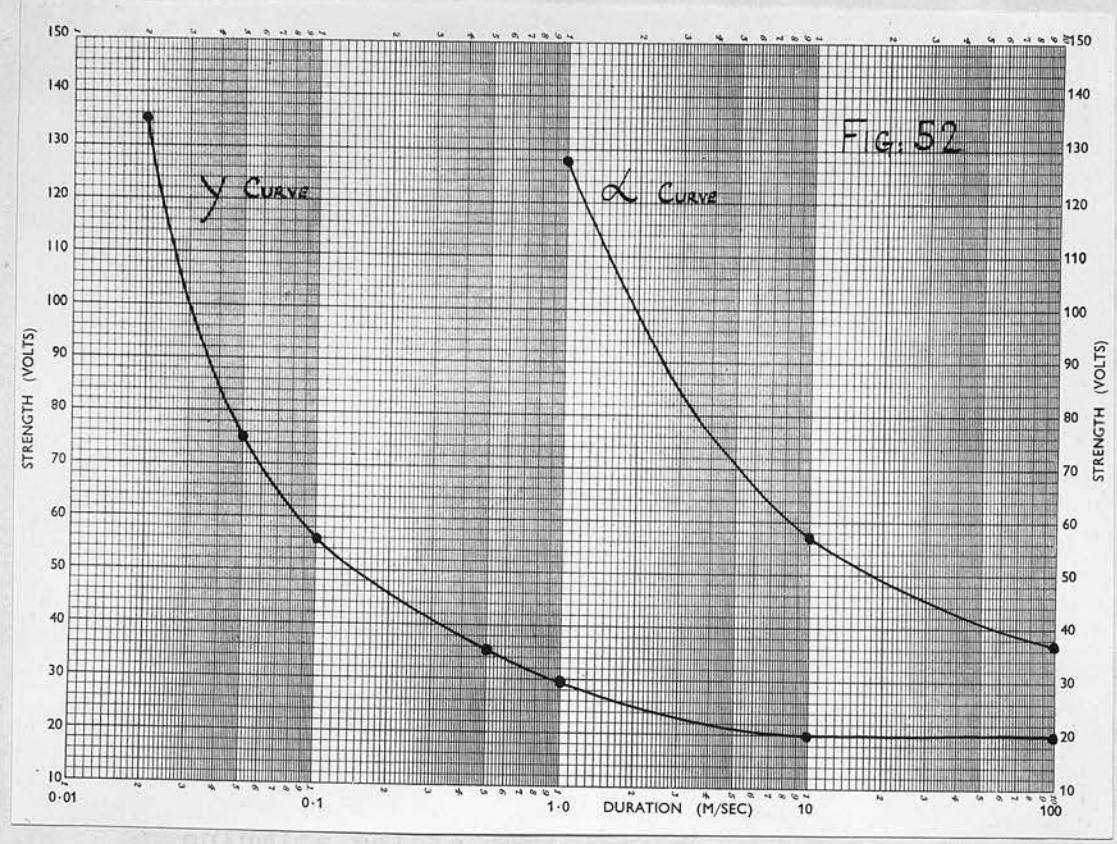
Rushton (1932) confirmed this observation for both muscle and nerve, and showed that the effect was less in the case of nerve than in muscle; this explained why Lapicque, using small electrodes, was unable to demonstrate discontinuities. Rushton also produced weighty evidence identifying the gamma excitability of muscle with nerve elements in the muscle.

Watts (1924) studying the strength-duration curve in the frog's sartorius showed that the alpha curve remained practically unchanged during degeneration; he also showed that curare abolished the gamma curve and lengthened the chronaxie of the beta curve, which he identified with the myoneural junction. Neither beta nor gamma curves were obtained after denervation. He believed that neuromuscular transmission was dependent on the integrity of the nerve and myoneural junction rather than an isochronism as suggested by Lapicque, and concludes:- "The increase in chronaxie observed in the human subject after denervation, is due, not to any alteration in the time constant of the muscle fibres, but merely to the point of incidence of the stimulus shifting from nerve to muscle".

Grundfest (1932) has made an elegant study of the strength-duration curves in a single nerve fibre muscle preparation in the frog. He showed that the chronaxie of both nerve and muscle varied with the size of the electrodes used, that that of muscle is affected more than that of nerve, but that neither is affected by curare; this confirms Watt's view of neuromuscular transmission. He also demonstrated complex curves by placing the exploring electrode on the point of junction of muscle and nerve.

Fig. 52 & 53.

The alpha and gamma curves



The theoretical direction of shift of discontinuities

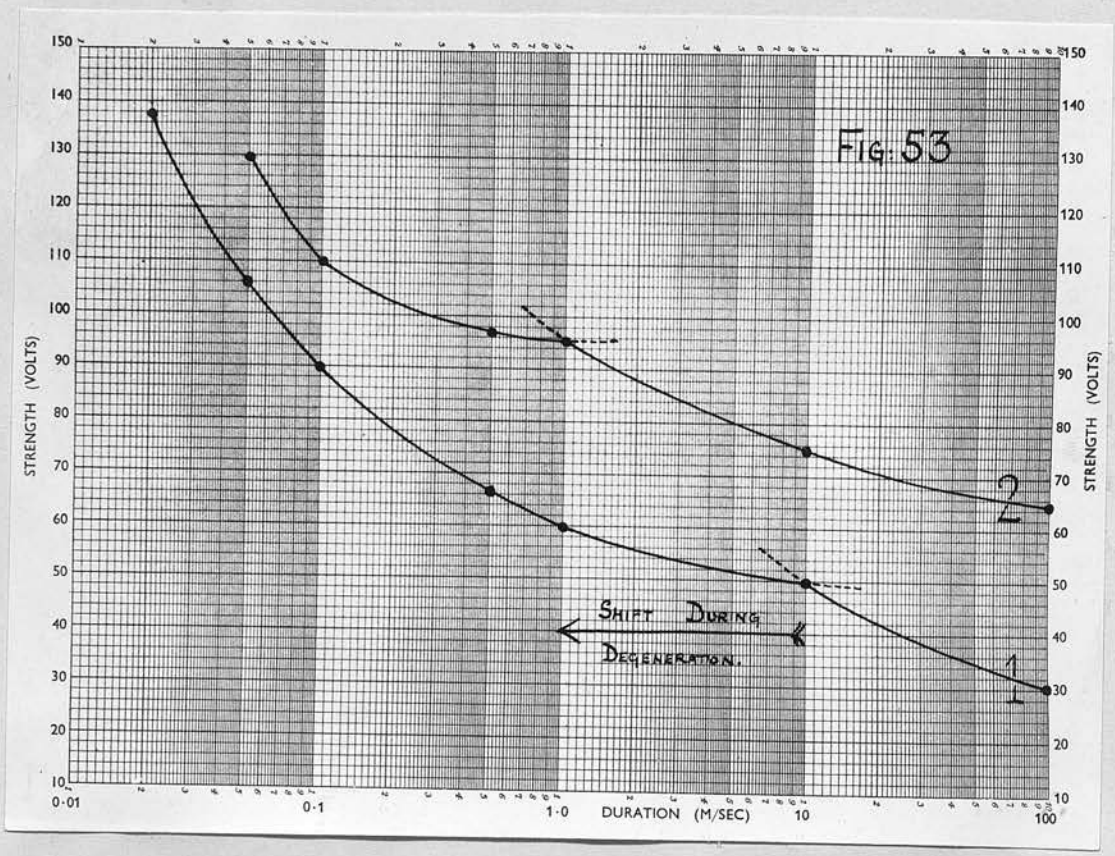


Fig.52 shows the curves of normal muscle (nerve elements) and denervated muscle (muscle fibres) on the same chart. Bauwens (1943) has described in some detail his concepts of the way in which these discontinuities appear during denervation, from the fusion of these two curves; if a muscle contains only a few denervated fibres, they will remain undetected in an examination of threshold stimuli, and at a voltage below their threshold they merely act as inert by-pass resistances which will entail the application of larger voltages to stimulate the normal muscle fibres. If higher voltages are used, the action of the paralysed fibres is masked by that of the normal fibres.

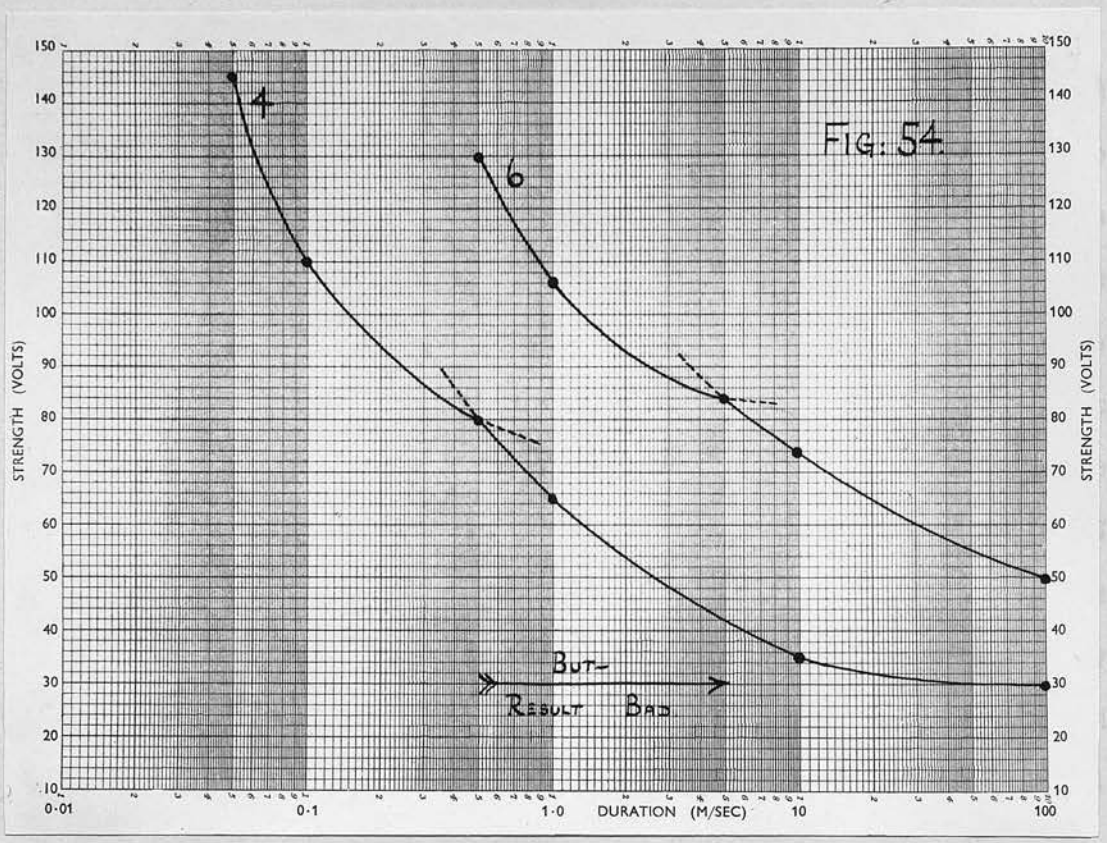
With a higher proportion of denervated fibres. the threshold of the gamma curve rises, and the alpha curve appears at the lower voltages and long durations of stimulus, and a discontinuity is produced. Fig.53,No.1.

With a still higher proportion of denervated fibres the threshold of the gamma curve rises still further, the alpha curve is more obvious, and the discontinuity appears at the shorter durations. Fig.53, No.2.

Thus, during denervation, the discontinuity should shift to the left and during recovery it should shift to the right. This may indeed occur in peripheral nerve injuries but in Poliomyelitis it does not.

Fig. 54 & 55.

Shift to the right in paralysed muscle in Poliomyelitis



Shift to the left in recovering muscle in Poliomyelitis

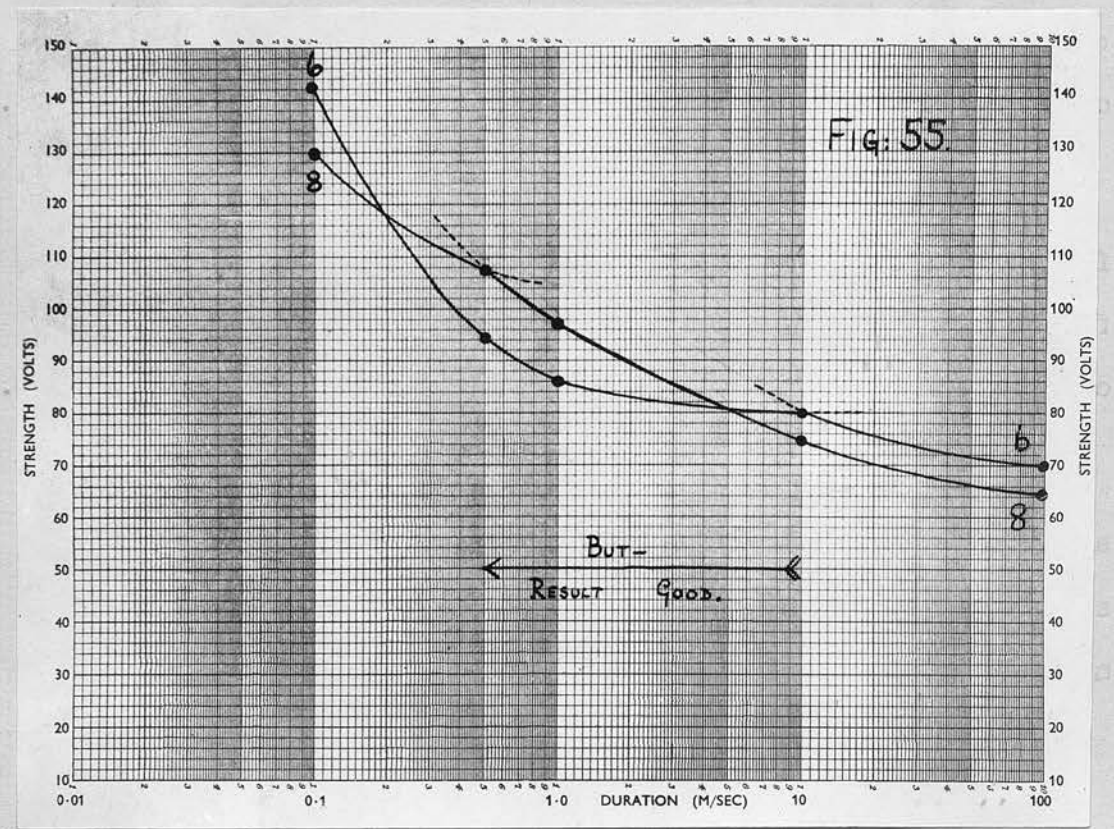


Fig.54 shows the curve from the right tibialis anterior of a severe case at the 4th and 6th weeks after the onset of the disease; the discontinuity shifted to the right but no recovery occurred in this muscle. The second curve is higher and its characteristics are less promising than that of the 4th week.

Fig.55 shows the opposite state of affairs. The curves from the right tibialis anterior of a case showing the discontinuity shifting to the left; but in this case a flicker of recovery was noted at the 7th week and ultimate recovery was quite good. It is thus apparent that the direction of shift of the discontinuity has no prognostic significance.

Discontinuities and speed of contraction

If discontinuities represent two modes of response in muscle, and if the response of muscle stimulated through its nerve is a rapid twitch and that of muscle stimulated direct a sluggish rise and fall, it should be possible to detect two speeds of contraction in muscles that give discontinuous curves; rapid at the short durations of stimulus and also at high voltages with long duration stimuli and slow at low voltages and long durations of stimulus.

The idea is illustrated in Fig.56 which shows a discontinuous curve obtained from the right tibialis anterior of a case six weeks after onset. In this case it was clinically obvious that there were indeed two different rates of contraction.

The phenomenon is however a difficult one to record. A rather crude attempt has been made using the tambour of a MacKenzie polygraph strapped over the tendon of the muscle at the ankle. The result is shown in Fig.57 which is taken from the same muscle that is illustrated in Fig.56.

The height of the excursion naturally depend on the vigour of the contraction and the strength of the stimulus; the rate of fall from the peak is rather slow in all cases as there is a good deal of inertia in the system of recording; the slope of the upstroke however gives an indication of the speed of the contraction, and does illustrate the point made.

The upper recording is at 0.5 M/S and shows a rapid twitch at a voltage little above the threshold.

The middle recording is at 100 M/S and shows a sluggish contraction at a voltage but little above the threshold.

The lower recording is again at 100 M/S and shows a rapid twitch at a voltage considerably above the threshold.

Mechanisms of recovery in Poliomyelitis.

Recovery in Poliomyelitis is a complex problem. The following table, modified from Bodian (1947) illus-

trates the various possibilities:-

TABLE 3

Anatomical site	Function in recovery
Motor cortex	Relearning of new pathways
Brain stem Spinal cord	Recovery of damaged cells Rerouting of internuncial paths
Motor nerves	Axonal regeneration Branching of intact fibres.
Motor end plates	Recovery
Muscle	Compensatory hypertrophy

The rerouting of the pathways in the brain stem and spinal cord, and the learning of the use of these pathways by the motor cortex need no further comment. These mechanisms offer a possible explanation for any incoordination of movement that may occur during recovery.

Positive evidence is now available for the supposition that reversible changes can occur in the anterior horn cells in the acute stages of the disease. Bodian (1947) has shown both in experimental and human material that intranuclear inclusions may be found in anterior horn cells of otherwise normal appearance in the subacute stages of the disease and the convalescent period. At an earlier period such inclusions are the accompaniment of severe cytoplasmic chromatolysis - an excellent indicator of axonal interruption - in the acute stages, and it therefore follows that such cells are recovering.

The possibility of axonal regeneration in Poliomyelitis has been mentioned by Bennett and Johnson

(1939) and Aegius, Bartolo, Coleiro and Seddon (1945), but it has never been more than an attractive theory. As we have seen, when a paralysed muscle recovers in Poliomyelitis the changes in the electrical reactions that accompany recovery are similar to those that occur in the peripheral nerve injuries as axons grow down and reinnervate the paralysed muscles. Admittedly, in Poliomyelitis the return of function is relatively early and since the rate of axonal regeneration is relatively slow (about 2 mm/day), regeneration cannot occur as a result of growth of the axons all the way from the cells to the periphery, but it is tempting to suppose that this may happen in a somewhat modified form.

Young (1945) has produced evidence to show that the axon behaves like a column of fluid the maintenance of whose length depends on the integrity of the enzyme systems within the anterior horn cells.

In Poliomyelitis the cell may be damaged without being destroyed, and it is conceivable that in its damaged state it is incapable of sustaining the whole length of axoplasm with the result that peripheral degeneration occurs; as the cell recovers, so that it can support a greater length of axoplasm, an outflow of axoplasm may occur, which, growing peripherally, will re-establish a normal peripheral connection, with the result that clinical recovery occurs.

It might well be that peripheral axonal degeneration and regeneration occurs and accounts for some of the paralysis which recovers. If, however, the process were as described above with actual degeneration of the axon, one would expect the electrical reactions to be precisely similar to those exibited after division of a peripheral nerve - the development of "R.D." in two to three weeks, and marked inexcitability of the muscle to electrical stimulation about the time of recovery. We have failed to show any such simple relationship.

In Poliomyelitis, as in peripheral nerve injuries, recovery in paralysed muscles is accompanied by inexcitability of the muscle. Moreover, the change in the strength-duration curve that preceed recovery are qualitatively similar to those described by Pollock as occurring after division of a peripheral nerve during the period of degeneration, but these changes are spread over a much longer period than three weeks.

This state of affairs could be explained on the assumption that there is a condition intermediate between axonotmesis and neurapraxia (Seddon 1944) - a condition in which the axon is intact but the electrical reactions are abnormal (unlike those in neurapraxia) - possibly due to a reversible peripheral degeneration of the medullary sheath.

Recovery of damaged motor end plates is mentioned in Table 3(p.68).Experimental evidence in favour of this possibility has been provided by Ibãnez (1944). He found in the guinea pig, that alterations were present in the motor end plates in every case of Poliomyelitis and that occasionally there were regenerative changes at a later stage. He also found that the nerves in the affected limb are only disturbed in their peripheral portions - additional evidence in favour of the possibility of peripheral axonal regeneration.

The possibility of branching of intact nerve fibres to supply paralysed muscle must be seriously considered. It is known to occur in peripheral nerve lesions in the rat and the rabbit (van Herreveld 1945 ; Weiss 1946) and may well play a part in recovery from human Poliomyelitis.

Time factors in Poliomyelitis

The importance of the 4th week after onset, in making a prognosis based on strength-duration curves, has been emphasised. Bodian (1947) has shown that chromatolysis of the anterior horn cells - the pathological indicator of axonal interruption - does not occur to a significant extent after this, so that presumably degenerative processes are at a maximum about this time.

This finding is difficult to reconcile with the observation that the reaction characteristic of dener-

vated muscle is not seen in some cases until 12 weeks after the onset of the disease. This leads one to suspect that the usual conception of Poliomyelitis as an explosive disease, in which the neuronal damage is maximal from the outset, may not be correct; a possible explanation of the late development of "R.D." would be a more slowly progressive lesion in which the initial neuronal damage is followed by further deterioration of the anterior horn cells. An alternative explanation would be that some cells may be badly damaged by the virus in the early stages, and only finally die as the result of some added insult such as early and excessive exercise of the muscles which they supply - i.e. excessive exercise might lead to actual degenerative changes. Further evidence is required before this question can be answered.

The corollary of this, the early development of "R.D." is also interesting, and Fig.24 illustrates a case in point. The data in this case are undoubtedly correct. The fully-developed curve characteristic of denervated muscle was present in four muscles nine days after the onset of the disease and four days after the onset of the paralysis. One explanation of this would be that the virus had entered and affected the lumbar spinal cord via the autonomic nerves from the intestine before any general signs appeared.

Electromyography and the Kenny concept

We have been unable to confirm the existence of "mental alienation" by this study of the electrical reactions in Poliomyelitis. Electromyographic studies carried out in America have, however, shown that some of Miss Kenny's other assertions may contain at least a germ of truth.

Incoordination of muscle activity has been demonstrated. Simultaneous contractions in agonist and antagonist have been demonstrated in Poliomyelitis (Watkins and Brazier 1943, 1944; Schwartz, Bouman and Smith 1944; Kohn 1945), in recovering peripheral nerve lesions (Watkins and Brazier 1943, 1944), and in cases of Poliomyelitis where the movements were painful (Brazier and Watkins 1944). Simultaneous contractions have also been demonstrated in the corresponding contralateral muscles in Poliomyelitis (Schwartz, Bouman and Smith 1944; Kohn 1945), and synchronous contractions have been demonstrated in different parts of the same muscle in a series of cases (Buchthal and Høncke 1944); asynchronism contractions in different parts of a muscle is the normal finding and asynchronism in a muscle with clinical signs of a severe paralysis is taken as a good prognostic omen.

Evidently, there is a disorder of reflex activity in Poliomyelitis which is more common than is clinically apparent or usually supposed. This may be

due to interruption of internuncial pathways followed by the relearning of new routes.

That this relearning can occur is demonstrated by an electromyographic study after tendon transplantation of the biceps to assist a weak quadriceps (Weiss and Brown 1941). The results are shown in the following table:-

TABLE 4

Period	Muscle	Time of action
Before op.	Quadriceps	Flexion and extension
	Biceps	Flexion only
Immediate pop.	"	No action
Later postop.	"	Flexion only
Convalescence	"	Flexion and extension
Late	"	Extension only, and liable to revert to flexion when fatigued.

Here, however, one is dealing with a whole muscle taking over a new function which is very different from conditions in Poliomyelitis.

Electromyography has also shown that muscle spasm occurs in Poliomyelitis. A normal muscle is silent at rest. Action potentials have been recorded by many observers in resting muscles in Poliomyelitis (Schwartz and Bouman 1942; Watkin and Brazier and Schwab 1943; 1944: Kohn 1945), and in some cases of polyneuritis (Brazier, Watkins and Schwab 1944). In both conditions their presence is correlated with weakness of the muscles

rather than with whatever tenderness may be present (Brazier, Watkins and Schwab 1944), hypersensitive to the chemical products of their contraction.

Normal muscle responds to stretching by the discharge of a few relatively small action potentials. In Poliomyelitis the response is of high voltage, and the discharge is persistent (Schwartz and Bouman 1942; Watkins, Brazier and Schwab 1943, 1944; Kohn 1945; Schwartz, Bouman and Smith 1944). This phenomenon has also been demonstrated in polyneuritis (Brazier, Watkins and Schwab 1944). It is not found in totally paralysed muscles in Poliomyelitis (Schwartz, Bouman 1942) but is widely present in apparently normal as well as partially paralysed muscles. It is most marked in those partially paralysed (Watkins, Brazier and Schwab 1943, 1944) and the response to stretching of these muscles is greater than that evoked by voluntary movement (Schwartz, Bouman and Smith 1944). It is similar to that found in muscles in the neighbourhood of a fracture (Watkins, Brazier and Schwab 1943).

The pathological site of origin of this spasm is difficult to determine; the lesions in fatal cases of Poliomyelitis are widespread and the spasm has usually disappeared at an earlier stage of the disease. Some writers (Schwartz, Bouman and Smith 1944) assume that the lesions responsible are those in the region of the cord that subserves the local reflex mechanisms; others

(Watkins, Brazier and Schwab 1943) attribute it to lesions of the meninges muscles, posterior roots or posterior horns.

Bodian (1946) considers these possibilities but concludes from his studies of human autopsy material that the lesions in these situations are not quantitatively sufficient to account for the phenomenon; he suggests that lesions in the motor cortex and the brain stem are responsible - by interfering with the normal inhibitor mechanisms. It has been shown (Lloyd 1941) that the vestibular centres are connected with motor neurons in the spinal cord, that stimulation of the bulbar reticular formation can have a generalised inhibitor effect on motor activity (Magoun 1944), and that spasticity can be produced by section of the reticulospinal tracts (Wagley 1945) in monkeys. Lesions of the internuncial neurons in the cord may also produce a similar effect since they form an integral part of higher inhibitor mechanism controlling the lower motor neurons.

In support of this theory, Bodian, using Rhesus monkeys infected with the Lansing strain of the virus by intracerebral inoculation, showed that spasticity was present when lesions were found in the motor cortex and the brain stem but before the lumbar cord showed any sign of damage.

Thus inco-ordination and spasm have been demonstrated both clinically and experimentally but out limited opportunities for observing the results of treatment on the lines laid down by Miss Kenny lead us to agree with the conclusions reached in the report of the Committee appointed by the Section of Orthopaedic Surgery of the American Medical Association for the investigation of this method of treatment (Ghormley 1944):-

> "The Kenny method of treatment does not prevent or even minimise the degree of permanent paralysis."

The Ritchie Stimulator

While in general the machine has been very satisfactory in use, it is suggested that a stimulus of longer duration than 100 milliseconds should be included in any subsequent model as it is clear that this does not give a true rheobase in many of the curves.

We have found that a modification of the circuit to halve the lowest rate of stimulation on the standard machine and give a rate of 0.75 cycles/sec simplifies the accurate determination of the end point.

The intermediate durations of stimulus - 0.5 M/S and 0.05 M/S are clearly important, and perhaps an additional duration of 5 M/S might be of advantage.

Ritchie (personal communication) is considering rearranging the durations of the stimulus so that they fall at regular intervals along the logarithmic scale of the abscissae of the graph. This would seem to be an excellent suggestion.

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SUMMARY

Poliomyelitis is an infectious disease due to a neurotropic virus; the prominent and crippling features of the disease, muscle weakness and paralysis are due to the action of this virus on the motorneuron cells of the brain stem and spinal cord.

The electrical reactions of the affected muscles may be of value in enabling a prognosis to be made earlier than can be done by clinical means.

In order to excite nerve or muscle, a stimulus must be of sufficient strength and duration, and its rate of change from zero to an effective height must be sufficiently rapid. Using an electronic stimulator, which delivers square-wave stimuli (Fig.1,p.9), one can neglect the last factor, and study the threshold strengths of stimulus required to produce a response from muscle at different durations. The result is a strength-duration curve (Fig.2, p.10), which is generally accepted as giving the best indication of the excitability of a muscle.

It is difficult to compare the whole of such a curve with other similar curves, and several indices have therefore been derived from the curves which may be used as a basis for such a comparison. Three such indices are the rheobase, which is defined as the threshold strength of a stimulus of infinite duration: the chronaxie

which is the threshold duration of a stimulus of twice rheobasic strength (Fig.2, p.10); and Lassalle's index which is obtained by multiplying the square of the rheobase by the chronaxie.

The interpretation of the electrical reactions of paralysed muscles in Poliomyelitis is very much more difficult than in peripheral nerve injuries since paralysis is often partial, and there is no means of knowing what the spatial arrangement of paralysed and functioning fibres is within the paralysed muscles - and therefore which group of fibres is near the examining electrode.

Strength-duration curves were performed at weekly intervals on a series of 30 patients with Poliomyelitis; variations in the characteristics of the curve were noted at various stages of the disease, and an attempt is made to correlate the findings with the course of the disease and to indicate how the strength-duration curve may serve as a guide to prognosis and treatment.

The normal strength-duration curve is smooth; the rheobase is relatively low, and the curve is flat at long durations of stimulus (Fig.5, p.19); the threshold at short duration of stimulus is higher, the curve rising steeply in this region, but at 0.5 M/S it is still but little higher than the rheobase. The chronaxie and Lassalle's index are both low; normal muscle responds

to all seven durations of stimulus on the Ritchie stimulator (which was used in this investigation), and its voluntary power is five - using the terminology recommended by the Peripheral Nerve Injury Committee of the Medical Research Council.

Discontinuities are found in a number of curves (Fig.6, p.20); they have been demonstrated in peripheral nerve injuries during degeneration and regeneration, but not in denervated or normal muscle.

The changes (described by previous workers) in strength-duration curves in peripheral nerve injuries are considered. The rheobase rises during degneration, is low in the denervated state - (we have been unable to confirm this in either peripheral nerve injuries or Poliomyelitis) and rises again with recovery, to remain high for a considerable period. The threshold for short duration stimuli rises during degeneration, is high in the denervated state, and falls during recovery. The chronaxie and Lassalle's index rise during degneration, are high in the denervated state, and fall abruptly with recovery only to rise again later before returning to a normal level. The number of stimuli of different durations which are effective in eliciting a response falls still further with recovery, and rises again to a normal value late in recovery.

The curve of unaffected muscle in Poliomyelitis is shown (Fig.13, p.28), and, after a consideration of the changes in the strength-duration curve of normal muscle than can be produced by altering its environment (which are very roughly +/- 10 volts), it is concluded that it is within normal limits.

Denervated muscle (Fig.21, p.36) only responds to long-duration stimuli; the rheobase, chronaxie and Lassalle's index are all higher than normal - the chronaxie markedly so - and the contraction of the muscle is more sluggish than normal.

Partially denervated muscle (Fig.22, p.37) gives a curve with characteristics intermediate between those of normal and denervated muscle.

The rate of development of these characteristic curves varies widely in different cases, and we have seen it from as early as nine days after the onset of the disease and four days after the onset of the paralysis to as late as 12 weeks after the onset of the disease.

These changes are summarised and correlated in a description of the changes which accompany the return of voluntary power in a muscle, and it is concluded that muscles that recover exhibit electrical reactions similar to those of innervated muscle except around the time of return of voluntary power - when the

muscle became relatively inexcitable and the curve obtained resembled that of denervated muscle. Chronaxie, rheobase, the threshold at short durations of stimulus and Lassalle's index all show fluctuations, but the indication of recovery is a fall of thse indices to a normal level following on a peak of many times normal value (Fig.26,p.41). This may occur before, after, or coincidental with the recovery of voluntary power, and to explain this it is suggested that conduction of an impulse and excitability to an electrical stimulus are two separate, if related properties of a nerve.

The curve of impending recovery in a muscle (Fig.28,p.43) is very similar to that of denervated muscle (Fig.29,p.43), the chronaxie of the former is usually higher, but this is not a reliable means to differentiation.

These changes are compared and contrasted with superficially similar changes that occur in paralysed muscles as they progress towards the final stable state of denervation; emphasis is laid on the fact that in muscle that recovers, although there are marked rises in the indices derived from the curves, between the peaks these indices fall to a relatively normal level, whereas in paralysed muscle there is a similar rise and fall in these indices but they never approach normal, and the changes are superimposed on an abnormal baseline.

Partially paralysed muscles (Fig.23, p.37) show changes similar to paralysed muscles, but those that we have examined have shown poor recovery, and it is possible that changes similar to those in recovering muscle would be found in partially paralysed muscles which make a good recovery.

It is suggested that a prognosis may be made on the result of the strength-duration curve at the 4th week after the onset of the disease, and reasons are given for believing that this is the optimum time for making such a prognosis.

Encouraging signs at the 4th week after onset are:-

Five or more effective stimuli (Fig.36,p.47).

Relatively high rheobase.

Relatively low chronaxie.

Lassalle's index and threshold for currents of short duration.

Signs on which a guarded prognosis should be made are:-

Four or less effective stimuli.

Relatively low rheobase.

Relatively high chronaxie.

Lassalle's index and threshold for short duration stimuli.

The interpretation of isolated curves at any time must be done with the greatest care since the actual figures obtained are largely dependent on the technique used.

Mere inspection of the curves at the 4th week is sufficient to distinguish many encouraging types but for certain intermediate curves, a more accurate method of differentiation is required. The following index is suggested for this purpose:-

$$1 = C(t - r) + t$$

where C = chronaxie

t = threshold at 0.5 M/S duration.

r = rheobase

This index gives values under 50 for normal curves; values under 150 indicate a good prognosis; values over 150 indicate that the prognosis should be guarded.

The effects of splintage and exercise on paralysed and weak muscles are considered in the light of the strength-duration curves obtained from such muscles, and it is concluded that excessive exercise may possibly adversely affect partially paralysed muscle, but that neither it nor splintage have any apparent effect on the strength-duration curves of recovering or totally paralysed muscles.

Miss Kenny has produced a "new" concept of the disease which lays emphasis on "spasm", "incoordination",

and "mental alienation" as being the primary features of the condition which will progress to paralysis if early and appropriate treatment is not undertaken.

The strength-duration curves in this series showed no evidence of the occurence of mental alienation. Spastic muscle gives a normal strength-duration curve so that one would not expect to obtain corroboration of spasm from this form of examination, but spasm - in the sense of a painful limitation of joint movement - was quite common in this series. Marked incoordination was found in one of the cases during recovery, but there was a distinct hysterical element in this case, and some doubt as to whether this was an organic or functional phenomenon. American work on electromyography in Poliomyelitis is summarised, and confirms the existence of Miss Kenny's methods of treatment do not lead us to believe that it offers anything that is not included in the more orthodox regimes.

The discussion contains conclusions on the value of an early prognosis in Poliomyelitis, the place of electrotherapy in treatment, the significance of the changes observed in the strength-duration curve in degeneration and recovery, the meaning of discontinuous strength-duration curves, the implications of variations in the time of development of the characteristic curves, and the mechanisms of recovery in Poliomyelitis.

The Ritchie stimulator is briefly discussed, and it is thought that a longer duration stimulus would be an asset, and that intermediate durations are valuable.

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CONCLUSION

1. A study of the strength-duration curves in Poliomyelitis is of value in making a prognosis and therefore in directing treatment.

2. Striking changes in the strength-duration curve accompanying denervation and recovery of voluntary power, but bear no regular temporal relationship to clinical recovery.

3. These changes are superficially similar to those described during degeneration and recovery in peripheral nerve injuries. In a muscle that will recover, fluctuations occur in rheobase, chronaxie, threshold at 0.5 M/S, Lassalle's index, and number of effective stimuli, but these changes are superimposed as a relatively normal baseline, and between the peaks, the values return to normal. In a denervated muscle, similar fluctuations occur, but are superimposed as an essentially abnormal baseline so that the values never fall quite to normal.

4. It is possible to make a prognosis on the results of the electrical reactions much earlier than can be done by clinical means. The optimum time for making such a prognosis is probably about the 4th week after the onset of the disease.

5. A good prognosis at the 4th week is based on a curve with the following characteristics:-

Five or more effective stimuli.
Relatively high rheobase (40).
Relatively low threshold for currents of short (0.5 M/S) duration (60).
Relatively low chronaxie (0.05).
Relatively low Lassalle's index (1000).

A bad prognosis is based on a curve with the opposite characteristics.

6. The index:- $1 = C(t - r) + t.$
where C = chronaxie
t = threshold at 0.5 M/S duration
r = rheobase

is of value in distinguishing the curves with intermediate characteristics at the 4th week. This index gives a value of:-

50 and under with normal curves.
150 and under with curves with a good prognosis.
150 and over with curves with a bad prognosis.

7. All these characteristics must be considered together in making a prognosis because other factors besides innervation are capable of altering the strength-duration curve ($\mp$ 10 volts) and the curve of impending recovery is very like that of denervated muscle.

8. These principles of prognosis may equally well be applied to partially paralysed as well as completely paralysed muscles.

9. Excessive exercise may have a deleterious effect on partially paralysed muscle.

10. Galvanism probably has no part to play in the treatment of Poliomyelitis.

11. There is no evidence that mental alienation as described by Miss Kenny occurs in the disease.

12. This investigation suggests that the following lines of further work might yield interesting results:-

a) Strength-duration curves in Poliomyelitis from the very earliest days.

b) Strength-duration curves as a large number of muscles at the 4th week after the onset of the paralysis to determine the reliability of the suggested index in making a prognosis.

c) Strength-duration curves of partially paralysed muscles to determine the value of the curves in prognosis and the effect of exercises and rest on the recovery in these muscles.

d) The cause of unusual discomfort which occurs in a certain number of cases subjected to this form of examination.

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